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TRANSMITTAL FORM

(to be used for all correspondence after initial filing)

Total Number of Pages in This Submission	103

Application Number	10/606,745
Filing Date	June 27, 2003
First Named Inventor	Peter Gluckman et al.
Art Unit	1654
Examiner Name	Jeffrey E. Russell
Attorney Docket Number	2014918.7046529001

ENCLOSURES ***(Check all that apply)***

- ☒ Fee Transmittal Form
  - ☐ Fee Attached
- ☐ Amendment/Reply
  - ☐ After Final
  - ☐ Affidavits/declaration(s)
- ☐ Extension of Time Request
- ☐ Express Abandonment Request
- ☐ Information Disclosure Statement
- ☐ Certified Copy of Priority Document(s)
- ☐ Reply to Missing Parts/ Incomplete Application
  - ☐ Reply to Missing Parts under 37 CFR1.52 or 1.53
- ☐ Drawing(s)
- ☐ Licensing-related Papers
- ☐ Petition
- ☐ Petition to Convert to a Provisional Application
- ☐ Power of Attorney, Revocation Change of Correspondence Address
- ☐ Terminal Disclaimer
- ☐ Request for Refund
- ☐ CD, Number of CD(s) _____
  - ☐ Landscape Table on CD
- ☐ After Allowance Communication to TC
- ☐ Appeal Communication to Board of Appeals and Interferences
- ☒ Appeal Communication to TC **(Appeal Notice, Brief, Reply Brief)**
- ☐ Proprietary Information
- ☐ Status Letter
- ☒ Other Enclosure(s) (please identify below):

U.S. Patent No. 5,714,460; copy of Decision on Motion from Interference No. 104,553 (Paper No. 111) and copy of Judgment from Interference No. 104,553 (Paper No. 116).

Remarks

SIGNATURE OF APPLICANT, ATTORNEY, OR AGENT

Firm Name	BINGHAM McCUTCHEN LLP		
Signature			
Printed Name	Erin M. Dunston		
Date	March 21, 2008	Reg. No.	51,147

CERTIFICATE OF TRANSMISSION/MAILING

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Signature			
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PTO/SB/17 (10-07)
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Effective on 12/08/2004.
Fees pursuant to the Consolidated Appropriations Act, 2005 (H.R. 4818).

FEE TRANSMITTAL for FY 2008

☐ Applicant claims small entity status. See 37 CFR 1.27

TOTAL AMOUNT OF PAYMENT ($) 510.00

Complete if Known	
Application Number	10/606,745
Filing Date	June 27, 2003
First Named Inventor	Peter Gluckman et al.
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METHOD OF PAYMENT (check all that apply)

☐ Check ☐ Credit Card ☐ Money Order ☐ None ☐ Other (please identify) : _____

☒ Deposit Account Deposit Account Number: 50-4047(704652-9001) Deposit Account Name: Bingham McCutchen LLP

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FEE CALCULATION

1. BASIC FILING, SEARCH, AND EXAMINATION FEES

	FILING FEES		SEARCH FEES		EXAMINATION FEES		
		Small Entity		Small Entity		Small Entity	
Application Type	Fee ($)	Fee($)	Fee($)	Fee($)	Fee($)	Fee($)	Fees Paid ($)
Utility	310	155	510	255	210	105	
Design	210	105	100	50	130	65	
Plant	210	105	310	155	160	80	
Reissue	310	155	510	255	620	310	
Provisional	210	105	0	0	0	0	

2. EXCESS CLAIM FEES

Fee Description	Fee ($)	Small Entity Fee ($)
Each claim over 20 (including Reissues)	50	25
Each independent claim over 3 (including Reissues)	210	105
Multiple dependent claims	370	185

Total Claims	Extra Claims	Fee($)	Fee Paid ($)
_____ -20 or HP=	_____	x _____	= _____

HP = highest number of total claims paid for, if greater than 20.

Indep. Claims	Extra Claims	Fee($)	Fee Paid ($)
_____ - 3 or HP=	_____	x _____	= _____

HP = highest number of independent claims paid for, if greater than 3.

Multiple Dependent Claims	
Fee ($)	Fee Paid ($)

3. APPLICATION SIZE FEE

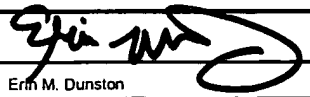
If the specification and drawings exceed 100 sheets of paper (excluding electronically filed sequence or computer listings under 37 CFR 1.52(e)), the application size fee due is $260 ($130 for small entity) for each additional 50 sheets or fraction thereof. See 35 U.S.C. 41(a)(1)(G) and 37 CFR 1.16(s).

Total Sheets	Extra Sheets	Number of each additional 50 or fraction thereof	Fee ($)	Fee Paid ($)
_____ - 100 =	_____ / 50 =	_____ (round up to a whole number) x	_____ =	

4. OTHER FEE(S)

	Fees Paid ($)
Non-English Specification, $130 fee (no small entity discount)	
Other (e.g., late filing surcharge) : Appeal Brief	510.00

SUBMITTED BY

Signature		Registration No. (Attorney/Agent)	51,147	Telephone	(202) 272-6000
Name (Print/Type)	Erin M. Dunston			Date	March 21, 2008

IN THE UNITED STATES PATENT AND TRADEMARK OFFICE

In re Patent Application of	)	**MAIL STOP APPEAL BRIEF-PATENTS**
Peter Gluckman *et al.*	)	
Application No.: 10/606,745	)	Group Art Unit: 1654
Filed: June 27, 2003	)	Examiner: Jeffrey E. RUSSELL
For: IGF-1 TO IMPROVE NEURAL OUTCOME	)	Confirmation No.: 5345

APPEAL BRIEF PURSUANT TO 37 C.F.R. § 41.37

MAIL STOP APPEAL BRIEF-PATENTS
Commissioner for Patents
P.O. Box 1450
Alexandria, Virginia 22313-1450

Sir:

Appellants hereby provide their Appeal Brief Pursuant To 37 C.F.R. § 41.37. This Appeal Brief is accompanied by the fee set forth in 37 C.F.R. § 41.20(b)(2). This Appeal Brief is being filed within one month of the Notice of Panel Decision from Pre-Appeal Brief Review, which was mailed on February 21, 2008.

TABLE OF CONTENTS

Page

I. Brief Procedural History

The instant application, U.S. Patent Application Serial No. 10/606,745 ("the '745 application"), is a reissue application for U.S. Patent No. 5,714,460 ("the '460 patent"). The '745 application was filed on June 27, 2003. The application that issued as the '460 patent, U.S. Patent Application Serial No. 08/460,365 ("the '365 application"), was filed on June 2, 1995. The '460 patent issued on February 3, 1998.

The '460 patent issued with fifteen (15) claims, including one (1) independent claim. Independent Claim 1 of the '460 patent reads:

> A method of treating neural damage suffered after a CNS insult affecting glia or other non-cholinergic cells in a mammal, comprising administering to the central nervous system of said mammal an effective amount of IGF-1 and/or a biologically active analogue of IGF-1.

Subsequent to the issuance of the '460 patent, Interference No. 104,553 ("the '553 Interference") was declared. At the conclusion of the '553 Interference, the Junior Party was Peter GLUCKMAN, who was involved in the '553 Interference based upon the '460 patent and U.S. Patent No. 5,861,373 ("the '373 patent"); and the Senior Party was Michael E. LEWIS *et al.*, who was involved in the '553 Interference based upon U.S. Patent Applications Serial Nos. 09/064,159 ("the '159 application") and 09/318,001 ("the '001 application").

The Decision on Motions in the '553 Interference was mailed on March 25, 2003. *See '553 Interference Paper No. 111.* Included within the Decision on Motions in the '553 Interference was the determination by the Board that Gluckman was entitled to the benefit of its earlier-filed New Zealand application. *Id. at Pages 38-39, 43.* Gluckman's New Zealand Patent Application No. 239211 was filed on August 1, 1991. *See id. at Page 15 n.11.*

The Board also determined that because Gluckman no longer had any patentable claims corresponding to the Count, judgment against Gluckman was appropriate. *Id. at Page 43.*

Judgment on priority as to Count 1 was awarded against Gluckman and the Board determined that Gluckman was not entitled to a patent containing Claims 1-15 of the '460 patent nor Claim 1 of the '373 patent. *'553 Interference Paper No. 116, Pages 1-2.*

II. Real Party In Interest

Pursuant to 37 C.F.R. § 41.37(c)(1)(i), the real party in interest for the '745 application is Genentech, Inc. *See Assignments recorded at Reel 015617, Frame 0156; Reel 015618, Frame 0583; and Reel 019110, Frame 0928.*

III. Related Appeals And Interferences

Pursuant to 37 C.F.R. § 41.37(c)(1)(ii), Appellants are aware of no "prior and pending appeals . . . or judicial proceedings . . . which may be related to, directly affect or be directly affected by or have a bearing on the Board's decision in this pending appeal."

However, Appellants are aware of a prior interference that is directly related to this application. Specifically, as discussed above in Section I, the '745 application is a reissue application for the '460 patent that was involved in the '553 Interference.

Accordingly, Appellants include, within Appendix 3 to this Brief, a copy of the Decisions on Motions (Paper No. 111) and a copy of the Judgment (Paper No. 116) from the '553 Interference.

IV. Status Of Claims

Pursuant to 37 C.F.R. § 41.37(c)(1)(iii), Appellants hereby provide a statement of the status of all the claims in the proceeding and identify those claims being appealed.

Claims 16, 28, and 64-77 are pending. *See, e.g., Office Action mailed July 20, 2007, Office Action Summary, Item 4.* Claims 16, 28, and 64-77 stand rejected. *Id. at Item 6.*

Claims 16, 28, and 64-77 stand rejected "as being based upon a defective reissue declaration under 35 U.S.C. 251." *Id. at Page 4, ¶ 6.* The Examiner has indicated that this rejection will be overcome if an "appropriate supplemental oath/declaration under 37 CFR 1.175(b)(1)" is received. *Id.*

Claims 16, 28, 66, 67, 72, and 73 stand rejected "under 35 U.S.C. 103 as being estopped on the merits by final judgment in Interference No. 104,5[5]3." *Id. at Pages 4-5, ¶ 8.* Similarly, Claims 16, 28, 66, 67, 72, and 73 stand rejected "under 35 U.S.C. 102(g) and/or 103 as being estopped on the merits by final judgment in Interference No. 104,5[5]3." *Id. at Pages 5-6, ¶ 9.*

Accordingly, the claims that have been substantively rejected are being appealed, *i.e.*, Claims 16, 28, 66, 67, 72, and 73. *See Appendix 1.*

V. Status Of Amendments

Pursuant to 37 C.F.R. § 41.37(c)(1)(iv), Appellants hereby state that no amendments have been filed subsequent to the rejection that was mailed on July 20, 2007.

VI. Summary Of Claimed Subject Matter

Pursuant to 37 C.F.R. § 41.37(c)(1)(v), Appellants hereby provide a "concise explanation of the subject matter defined in each of the independent claims involved in the appeal, which shall refer to the specification by page and line number, and to the drawing, if any, by reference character."

Of the claims appealed, there are two (2) independent claims: Claims 16 and 28.

Claim 16 reads:

> A method of treating glial cells damaged from CNS injury, wherein said CNS injury predominantly affects glia, comprising administering to the CNS of a mammal in need thereof, an effective amount of IGF-1.

Support for Claim 16 can be found throughout the '460 patent, and at least in the Abstract and at Column 1, Lines 47-61 of the '460 patent.

Claim 28 reads:

> A method of treating glial cells damaged from CNS injury, wherein said CNS injury predominantly affects glia, comprising administering to the CNS of a mammal in need thereof, an effective amount of a biological analog of IGF-1, wherein said analog is selected from the group consisting of naturally-occurring analogs, IGF-2, and des 1-3 IGF-1.

Support for Claim 28 can be found throughout the '460 patent, and at least in the Abstract and at Column 1, Lines 47-61; Column 3, Lines 35-36; and Column 3, Lines 43-35.

VII. Grounds Of Rejection To Be Reviewed On Appeal

Pursuant to 37 C.F.R. § 41.37(c)(1)(vi), Appellants hereby provide a "concise statement of each ground of rejection presented for review."

There are two related grounds of rejection presented for review: (1) Whether Claims 16, 28, 66, 67, 72, and 73 are estopped on the merits by final judgment in the '553 Interference under 35 U.S.C. § 103; and (2) Whether Claims 16, 28, 66, 67, 72, and 73 are estopped on the merits by final judgment in the '553 Interference under 35 U.S.C. §§ 102(g) and/or 103.

VIII. Argument

Pursuant to 37 C.F.R. § 41.37(c)(1)(vii), Appellants hereby provide their contentions "with respect to each ground of rejection presented for review . . . and the basis therefor, with citations of the statutes, regulations, authorities, and parts of the record relied on." Each ground of rejection will be treated under a separate heading.

A. Rejection Of Claims 16, 28, 66, 67, 72, and 73 Due To Purported Estoppel On The Merits By Final Judgment In The '553 Interference Under 35 U.S.C. § 103

Claims 16, 28, 66, 67, 72, and 73 stand rejected "under 35 U.S.C. 103 as being estopped on the merits by final judgment in Interference No. 104,5[5]3." *Office Action mailed July 20, 2007, at Pages 4-5, ¶ 8.* These rejections are respectfully traversed.

1. Claim 16

According to the Examiner:

> Claims 16, 28, 66, 67, 72, and 73 are rejected under 35 U.S.C. 103 as being estopped on the merits by final judgment in Interference No. 104,5[5]3. In the section of the interference count which corresponds to claim 1 of U.S. Patent No. 5,714,460, damaged glia or other non-cholinergic cells are treated with IGF-1 or biologically active analogues thereof. The interference count does not recite that the CNS injury predominantly affects glia. It would have been obvious to one of ordinary skill in the art at the time Applicants' invention was made to treat glial cells damaged by CNS injury which predominantly affects glia, because the interference count specifically recites that injured glia cells are to be treated, and the lack of injury to other types of cells would not have been expected to interfere with the ability of IGF-1 and its analogues to treat injured glial cells. With respect to claims 66, 67, 72, and 73, these claims recite the same or broader limitations as are recited in claims 8-9 of the '460 patent, which were designated as corresponding to the count. Gluckman filed a motion in the interference contesting the designation of this claim as corresponding to the count, which motion was denied (see pages 24-26 of the Decision on Motions). Accordingly, claims 66, 67, 72, and 73 are deemed obvious over that section of the count which corresponds to claim 1 of U.S. Patent No. 5,714,460. See 37 CFR 41.127(a) and MPEP 2308.03, Examples 2 and 3 (Rev. 4, October 2005).

> In the paper titled "Notice Under 37 C.F.R. § 1/178(b)" filed June 27, 2003, Applicants refer to footnote 17 of the Decision on Motions in the interference as indicating that Applicants would not be estopped from pursuing in a reissue application narrower claims that would not have been obvious in view of the lost count. As indicated above, the current reissue claims remain obvious over the lost count.

Office Action mailed July 20, 2007, Pages 4-5, ¶ 8.

Appellants respectfully disagree. During the course of the '553 Interference, the Count was Gluckman Claim 1 from the '460 patent OR Gluckman Claim 1 from the '373 patent OR Lewis Claim 129 from U.S. Patent Application Serial No. 09/064,159 ("the '159 application") OR Lewis Claim 135 of U.S. Patent Application Serial No. 09/318,001 ("the '001 application"). *Decision on Motion in Interference 104,553, Paper No. 111, ¶¶ 1-5, 12, 13.* Accordingly, the Count was:

Claim 1 of the '460 patent: A method of treating neural damage suffered after a CNS insult affecting glia or other non-cholinergic cells in a mammal, comprising administering to the central nervous system of said mammal an effective amount of IGF-1 and/or a biologically active analogue of IGF-1. **OR**

Claim 1 of the '373 patent: A method of treating neural damage suffered after a CNS insult affecting glia or other non-cholinergic cells in a mammal, comprising administering to the central nervous system of said mammal a medicament comprising an effective amount of IGF-1 and/or a biologically active analog of IGF-1. **OR**

Claim 129 of the '159 application: A method of treating a mammal subject to a neurological disease comprising administering to said mammal an

amount of insulin-like growth factor I effective to enhance the survival of non-mitotic neuronal cells at risk of dying.

OR

Claim 135 of the '001 application: A method of enhancing the survival of CNS neuronal cells in a mammal, said cells being at risk of dying caused by injury or epilepsy, which method comprises administering to said mammal an effective amount of IGF-1.

During the '553 Interference, the Count was given its broadest reasonable construction. *Decision on Motion in Interference 104,553, Paper No. 111, Page 40.* The Board noted that the "broadest reasonable construction of Gluckman's 460 claim 1 and 373 claim 1 is that the treatment must be for a CNS insult 'affecting' glia or other non-cholinergic cells, but may also affect cholinergic neurons." *Id.* The finding by the Board that Gluckman's claimed method could unintentionally also treat cholinergic cells was the basis for its denial of Gluckman's motion, referenced in the quote above by the Examiner.

In contrast, Appellants' Claim 16, as well as Claims 28, 66, 67, 72, and 73, are directed to the treatment of a CNS injury wherein the CNS injury *predominantly affects glia*. As noted in the Decision on Motions in the '553 Interference, "neither party discloses a targeted treatment in a patient of one type of CNS cell to the exclusion of other CNS cells." *Decision on Motion in Interference 104,553, Paper No. 111, ¶ 26.* The Lewis applications involved in the '553 Interference neither taught nor claimed treating CNS insults affecting predominantly glia. Gluckman could not add such narrower claims into the '553 Interference because they would have been non-interfering claims, and parties are precluded in an

interference from adding non-interfering claims. *See discussion below*. As such, Gluckman should not now be estopped from adding such non-interfering claims to this reissue application. Moreover, claims which could not have been added to an interference cannot be estopped by a decision in the '553 Interference. The Board noted this in footnote 17, at Page 23, of the Decision on Motions. *Decision on Motion in Interference 104,553, Paper No. 111, Page 23 n.17*. Appellants respectfully submit that the Count from the '553 Interference does not render obvious Appellants' Claims 16, 28, 66, 67, 72, and 73 which now specifically require that the CNS injury predominantly affects glia.

In addition, Appellants respectfully submit that a *prima facie* case of obviousness has not been made against Claim 16. Obviousness is a question of law, based upon several factual inquiries (known as "the *Graham* factors"), including determining (1) the scope and content of the prior art; (2) the level of ordinary skill in the art; (3) the differences between the claimed invention and the prior art; and (4) whether the differences are such that the claimed invention as a whole would have been obvious to one of ordinary skill in the art at the time the invention was made. *See Ruiz v. A.B. Chance Co.*, 234 F.3d 654, 660 (Fed. Cir. 2000) (citing *Graham v. John Deere Co.*, 383 U.S. 1, 17-18 (1966)).

When making an obviousness rejection, Examiners are instructed to "ensure that the written record includes findings of fact concerning the state of the art and the teachings of the references applied. . . . Factual findings made by Office personnel are the necessary underpinnings to establish obviousness. . . . Office personnel must provide an explanation to support an obviousness rejection under 35 U.S.C. 103. 35 U.S.C. 132 requires that the applicant be notified of the reasons for the rejection of the claim so that he or she can decide how best to proceed. . . . In short, the focus when making a determination of obviousness should be on what

a person of ordinary skill in the pertinent art would have known at the time of the invention, and on what such a person would have reasonably expected to have been able to do in view of that knowledge." 72(195) Fed. Reg. 57526, at 57527 (Oct. 10, 2007). Examiners bear the initial burden of factually supporting any *prima facie* conclusion of obviousness and if such a case is not made, "the applicant is under no obligation to submit evidence of nonobviousness." *M.P.E.P. § 2142* (noting also that the "key to supporting any rejection under 35 U.S.C. 103 is the clear articulation of the reason(s) why the claimed invention would have been obvious").

Appellants respectfully submit that a *prima facie* case of obviousness as to Claim 16 has not been made. There are no factual findings of record for the '745 application regarding the *Graham* factors. That is, there are no findings regarding: (1) the scope and content of the prior art; (2) the level of ordinary skill in the art; (3) the differences between the claimed invention and the prior art; and (4) whether the differences are such that the claimed invention as a whole would have been obvious to one of ordinary skill in the art at the time the invention was made. *See Ruiz*, 234 F.3d at 660. There is no basis, other than the Examiner's own opinion, for the assertion that "It would have been obvious to one of ordinary skill in the art at the time Applicants' invention was made to treat glial cells damaged by CNS injury which predominantly affects glia, because the interference count specifically recites that injured glia cells are to be treated, and the lack of injury to other types of cells would not have been expected to interfere with the ability of IGF-1 and its analogues to treat injured glial cells." *Office Action mailed July 20, 2007, Pages 4-5, ¶ 8.*

Moreover, a party cannot seek to add a claim to an application in interference and request that it be designated as not corresponding to the Count. *L'Esperance v. Nishimoto*, 18 U.S.P.Q.2d 1534 (Bd. Pat. App. & Int. 1991). The Decision on Motions from the '553

Interference notes that because Gluckman could *not* add narrower claims, such as Claim 16, Gluckman would *not be estopped* from filing a reissue application seeking narrower claims that would not have been obvious in view of the subject matter of the lost count to the extent Gluckman's specification supports such claims. *Decision on Motions in Interference 104,553, Paper No. 111, Page 23 n.17.* Claim 16 is narrower than the claims involved in the '553 Interference at least because it specifies that the CNS injury predominantly affects glia. Thus, interference estoppel does *not* attach because Appellants were not able to add such a claim during the course of the '553 Interference.

a. Claim 66

Claim 66 depends from Claim 16, but specifies that the CNS injury is a demyelinating disorder. As such, Claim 66 includes all of the limitations of Claim 16, and adds another limitation. Appellants respectfully submit that the rejection of Claim 66 under 35 U.S.C. § 103 due to purported estoppel on the merits by final judgment in the '553 Interference is in error at least for the same reasons as that rejection is in error for Claim 16.

Further and as with Claim 16, a *prima facie* case of obviousness has not been made out against Claim 66. There are no factual findings of record for the '745 application regarding the *Graham* factors as they pertain to Claim 66. *See Section VIII.A.I, above.* There is no basis, other than the Examiner's own opinion, as to the purported obviousness of Claim 66. *See id.*

Again similar to the situation with Claim 16, Claim 66 is narrower than the claims involved in the '553 interference at least because it specifies that the CNS injury predominantly affects glia, and further specifies that the CNS injury is a demyelinating disorder. Because Appellants could not have added Claim 66 during the interference, interference estoppel does not attach to Claim 66.

b. Claim 67

Claim 67 depends from Claim 16, but specifies that the CNS injury is multiple sclerosis. As such, Claim 67 includes all of the limitations of Claim 16, and adds another limitation. Appellants respectfully submit that the rejection of Claim 67 under 35 U.S.C. § 103 due to purported estoppel on the merits by final judgment in the '553 Interference is in error at least for the same reasons as that rejection is in error for Claim 16.

Further and as with Claim 16, a *prima facie* case of obviousness has not been made out against Claim 67. There are no factual findings of record for the '745 application regarding the *Graham* factors as they pertain to Claim 67. *See Section VIII.A.I, above.* There is no basis, other than the Examiner's own opinion, as to the purported obviousness of Claim 67. *See id.*

Again similar to the situation with Claim 16, Claim 67 is narrower than the claims involved in the '553 interference at least because it specifies that the CNS injury predominantly affects glia, and further specifies that the CNS injury is multiple sclerosis. Because Appellants could not have added Claim 67 during the interference, interference estoppel does not attach to Claim 66.

2. Claim 28

Claim 28 is similar to Claim 16, yet specifies that instead of administering IGF-1, *per se*, an effective amount of a biological analog of IGF-1 is administered, wherein that analog is a naturally-occurring analog, IGF-2, or des 1-3 IGF-1. Like Claim 16, Claim 28 is directed to the treatment of CNS injury wherein the CNS injury *predominantly affects glia.* As noted in the Decision on Motions in the '553 Interference, "neither party discloses a targeted treatment in a patient of one type of CNS cell to the exclusion of other CNS cells." *Decision on Motion in Interference 104,553, Paper No. 111, ¶ 26.* Appellants respectfully submit that the Count from

the '553 Interference does not render obvious Appellants' Claim 28, which specifically requires that the CNS injury predominantly affects glia.

In addition, Appellants respectfully submit that a *prima facie* case of obviousness has not been made against Claim 28. Obviousness is a question of law, based upon the *Graham* factors, enumerated above. Appellants respectfully submit that a *prima facie* case of obviousness as to Claim 28 has not been made. There are no factual findings of record for the '745 application regarding the *Graham* factors. The record lacks the "[f]actual findings made by Office personnel [that] are the necessary underpinnings to establish obviousness." 72(195) Fed. Reg. 57526, at 57527 (Oct. 10, 2007). There is no basis, other than the Examiner's own opinion, for the assertion that "It would have been obvious to one of ordinary skill in the art at the time Applicants' invention was made to treat glial cells damaged by CNS injury which predominantly affects glia, because the interference count specifically recites that injured glia cells are to be treated, and the lack of injury to other types of cells would not have been expected to interfere with the ability of IGF-1 and its analogues to treat injured glial cells." *Office Action mailed July 20, 2007, Pages 4-5, ¶ 8.*

Moreover, a party cannot seek to add a claim to an application in interference and request that it be designated as not corresponding to the Count. *L'Esperance v. Nishimoto*, 18 U.S.P.Q.2d 1534 (Bd. Pat. App. & Int. 1991). The Decision on Motions from the '553 Interference notes that because Gluckman could *not* add narrower claims, such as Claim 28, Gluckman would *not be estopped* from filing a reissue application seeking narrower claims that would not have been obvious in view of the subject matter of the lost count to the extent Gluckman's specification supports such claims. *Decision on Motions in Interference 104,553, Paper No. 111, Page 23, Page 23, n.17.* Claim 28 is narrower than the claims involved in the

'553 Interference at least because it specifies that the CNS injury predominantly affects glia and further specifies that instead of administering IGF-1, *per se*, an effective amount of a biological analog of IGF-1 is administered, wherein that analog is a naturally-occurring analog, IGF-2, or des 1-3 IGF-1. Thus, interference estoppel does *not* attach because Appellants were not able to add such a claim during the course of the '553 Interference.

a. Claim 72

Claim 72 depends from Claim 28, but specifies that the CNS injury is a demyelinating disorder. As such, Claim 72 includes all of the limitations of Claim 28, and adds another limitation. Appellants respectfully submit that the rejection of Claim 72 under 35 U.S.C. § 103 due to purported estoppel on the merits by final judgment in the '553 Interference is in error at least for the same reasons as that rejection is in error for Claim 28.

Further and as with Claim 28, a *prima facie* case of obviousness has not been made out against Claim 72. There are no factual findings of record for the '745 application regarding the *Graham* factors as they pertain to Claim 72. *See Sections VIII.A.I and VIII.A.2, above*. There is no basis, other than the Examiner's own opinion, as to the purported obviousness of Claim 72. *See id.*

Again similar to the situation with Claim 28, Claim 72 is narrower than the claims involved in the '553 interference at least because it specifies that the CNS injury predominantly affects glia, and further specifies that the CNS injury is a demyelinating disorder. Because Appellants could not have added Claim 72 during the interference, interference estoppel does not attach to Claim 72.

b. Claim 73

Claim 73 depends from Claim 28, but specifies that the CNS injury is multiple sclerosis. As such, Claim 73 includes all of the limitations of Claim 28, and adds another limitation. Appellants respectfully submit that the rejection of Claim 73 under 35 U.S.C. § 103 due to purported estoppel on the merits by final judgment in the '553 Interference is in error at least for the same reasons as that rejection is in error for Claim 28.

Further and as with Claim 28, a *prima facie* case of obviousness has not been made out against Claim 73. There are no factual findings of record for the '745 application regarding the *Graham* factors as they pertain to Claim 73. *See Sections VIII.A.I and VIII.A.2, above.* There is no basis, other than the Examiner's own opinion, as to the purported obviousness of Claim 73. *See id.*

Again similar to the situation with Claim 28, Claim 73 is narrower than the claims involved in the '553 interference at least because it specifies that the CNS injury predominantly affects glia, and further specifies that the CNS injury is multiple sclerosis. Because Appellants could not have added Claim 73 during the interference, interference estoppel does not attach to Claim 73.

3. Conclusion

Appellants respectfully assert that a *prima facie* case of obviousness has not been made out against any of Claims 16, 28, 66, 67, 72, and 73. Moreover, Claims 16, 28, 66, 67, 72, and 73 are narrower than the claims involved in the '553 Interference and, as such, Appellants could not have added them to the '553 Interference. Therefore, interference estoppel should not attach to Claims 16, 28, 66, 67, 72, and 73.

In view of the foregoing, Appellants respectfully request withdrawal of the rejection under 35 U.S.C. § 103 of Claims 16, 28, 66, 67, 72, and 73 due to purported estoppel on the merits by final judgment in the '553 Interference.

B. Rejection of Claims 16, 28, 66, 67, 72, and 73 Due To Purported Estoppel On The Merits By Final Judgment In The '553 Interference Under 35 U.S.C. §§ 102(g) And/Or 103

Claims 16, 28, 66, 67, 72, and 73 stand rejected "under 35 U.S.C. 102(g) and/or 103 as being estopped on the merits by final judgment in Interference No. 104,5[5]3." *Office Action mailed July 20, 2007, at Pages 5-6, ¶ 9.* These rejections are respectfully traversed.

According to the Examiner:

> Claims 16, 28, 66, 67, 72, and 73 are rejected under 35 U.S.C. 102(g) and/or 103 as being estopped on the merits by final judgment in Interference No. 104,5[5]3. In the section of the interference count which corresponds to claim 1 of U.S. Patent No. 5,714,460, damaged glia or other non-cholinergic cells are treated with IGF-1 or biologically active analogues thereof. With respect to claims 16, 28, 66, 67, 72, and 73, these claims recite the same or broader limitations as is recited in claims 8-9 of the '460 patent, which were designated as corresponding to the count. Gluckman filed a motion in the interference contesting the designation of this claim as corresponding to the count, which motion was denied (see pages 24-26 of the Decision On Motions). Further, Applicants' specification at column 1, lines 44-46, acknowledges that multiple sclerosis is a disease of the CNS which causes the loss of oligodendrocytes (which are a type of glia cells). This section of Applicants' specification is quoted in section [19] of the Decision on Motions. See also section [93] of the Decision on Motions. Accordingly, the count, which suggests treating glial cells which are injured by multiple sclerosis, also suggests the broader limitation of treating glial cells damaged by CNS injury which predominantly affects glia. Claims 16, 28, 66, 67, 72, and 73 are deemed obvious over that section of the count which corresponds to claim 1 of U.S. Patent No. 5,714,460. See 37 CFR 41.127(a) and MPEP 2308.03, Examples 2 and 3 (Rev. 4, October 2005). In the paper titled "Notice Under 37 C.F.R. §1.178(b)" filed June 27, 2003, Applicants refer to footnote 17 of the Decision on Motions in the interference as indicating that Applicants would not be estopped from pursuing in a reissue application narrower claims that would have not been obvious in view of the lost count. However, the basis for this approach is that the reissue

> claims must be nonobvious over the lost count. As indicated above, the current reissue claims remain obvious over (or even anticipated by) the lost count.

Office Action mailed July 20, 2007, Pages 5-6, ¶ 9. Appellants respectfully disagree.

1. Purported Anticipation Under 35 U.S.C. § 102(g)

Appellants note that "[i]nvalidity based on 'anticipation' [under 35 U.S.C. § 102] requires that the invention is not in fact new." *Verve, LLC v. Crane Cams, Inc.*, 311 F.3d 1116, 1120 (Fed. Cir. 2002) (quoting *Hoover Group, Inc. v. Custom Metalcraft, Inc.*, 66 F.3d 299, 302 (Fed. Cir. 1995)). "A single reference must describe the claimed invention with sufficient precision and detail to establish that the subject matter existed in the prior art." *Verve*, 311 F.3d at 1120 (citing *In re Spada*, 911 F.2d 705, 708 (Fed. Cir. 1990)). Put differently, "[a] claim is anticipated only if each and every element as set forth in the claim is found, either expressly or inherently described, in a single prior art reference." *Verdegaal Bros. v. Union Oil Co. of California*, 814 F.2d 628, 631 (Fed. Cir. 1987).

a. Claim 16

Appellants assert that Claim 16 is not estopped on the merits under 35 U.S.C. § 102(g) by the final judgment in the '553 Interference at least because each and every element of Claim 16 was not disclosed or involved in the '553 Interference. During the course of the '553 Interference, the Count was Gluckman Claim 1 from the '460 patent OR Gluckman Claim 1 from the '373 patent OR Lewis Claim 129 from U.S. Patent Application Serial No. 09/064,159 OR Lewis Claim 135 of U.S. Patent Application Serial No. 09/318,001. *Decision on Motion in Interference 104,553, Paper No. 111, ¶¶ 1-5, 12, 13.* Accordingly, the Count was:

Claim 1 of the '460 patent: A method of treating neural damage suffered after a CNS insult affecting glia or other non-cholinergic cells in a mammal,

comprising administering to the central nervous system of said mammal an effective amount of IGF-1 and/or a biologically active analogue of IGF-1. **OR**

Claim 1 of the '373 patent: A method of treating neural damage suffered after a CNS insult affecting glia or other non-cholinergic cells in a mammal, comprising administering to the central nervous system of said mammal a medicament comprising an effective amount of IGF-1 and/or a biologically active analog of IGF-1. **OR**

Claim 129 of the '159 application: A method of treating a mammal subject to a neurological disease comprising administering to said mammal an amount of insulin-like growth factor I effective to enhance the survival of non-mitotic neuronal cells at risk of dying.

OR

Claim 135 of the '001 application: A method of enhancing the survival of CNS neuronal cells in a mammal, said cells being at risk of dying caused by injury or epilepsy, which method comprises administering to said mammal an effective amount of IGF-1.

During the '553 Interference, the Count was given its broadest reasonable construction. *Decision on Motion in Interference 104,553, Paper No. 111, Page 40.* The Board noted that the "broadest reasonable construction of Gluckman's 460 claim 1 and 373 claim 1 is that the treatment must be for a CNS insult 'affecting' glia or other non-cholinergic cells, but may also affect cholinergic neurons." *Id.* The finding by the Board that Gluckman's claimed method

could unintentionally also treat cholinergic cells was the basis for its denial of Gluckman's motion, referenced in the quote above by the Examiner.

In contrast, Appellants' Claim 16, as well as Claims 28, 66, 67, 72, and 73, are directed to the treatment of a CNS injury wherein the CNS injury *predominantly affects glia*. As noted in the Decision on Motions in the '553 Interference, "neither party discloses a targeted treatment in a patient of one type of CNS cell to the exclusion of other CNS cells." *Decision on Motion in Interference 104,553, Paper No. 111, ¶ 26.* The Lewis applications involved in the '553 Interference neither taught nor claimed treating CNS insults affecting predominantly glia. Gluckman could not add such narrower claims into the '553 Interference because they would have been non-interfering claims, and parties are precluded in an interference from adding non-interfering claims. *See discussion below.* As such, Gluckman should not now be estopped from adding such non-interfering claims to this reissue application. Moreover, claims which could not have been added to an interference cannot be estopped by a decision in the '553 Interference. The Board noted this in footnote 17, at Page 23, of the Decision on Motions. *Decision on Motion in Interference 104,553, Paper No. 111, Page 23 n.17.*

In contrast, Appellants' Claim 16, as well as Claims 28, 66, 67, 72, and 73, are directed to the treatment of a CNS injury wherein the CNS injury *predominantly affects glia*. As noted in the Decision on Motions in the '553 Interference, "neither party discloses a targeted treatment in a patient of one type of CNS cell to the exclusion of other CNS cells." *Decision on Motion in Interference 104,553, Paper No. 111, ¶ 26.* Appellants respectfully submit that the Count from the '553 Interference does not anticipate Claim16 (nor does it anticipate Claims 28, 66, 67, 72, and 73) because it fails to disclose each and every element of

Claim 16 (and Claims 28, 66, 67, 72, and 73), *e.g.*, that the CNS injury predominantly affects glia.

i. Claim 66

Claim 66 depends from Claim 16, but specifies that the CNS injury is a demyelinating disorder. As such, Claim 66 includes all of the limitations of Claim 16, yet adds another limitation. Appellants respectfully submit that the anticipation rejection of Claim 66 under 35 U.S.C. § 102(g) due to purported estoppel on the merits by final judgment in the '553 Interference is in error at least for the same reasons as that rejection is in error for Claim 16. In addition, Claim 66's additional limitation, that the CNS injury is a demyelinating disorder, is absent from the '553 Interference.

ii. Claim 67

Claim 67 depends from Claim 16, but specifies that the CNS injury is multiple sclerosis. As such, Claim 67 includes all of the limitations of Claim 16, yet adds another limitation. Appellants respectfully submit that the anticipation rejection of Claim 67 under 35 U.S.C. § 102(g) due to purported estoppel on the merits by final judgment in the '553 Interference is in error at least for the same reasons as that rejection is in error for Claim 16. In addition, Claim 67's additional limitation, that the CNS injury is multiple sclerosis, is absent from the '553 Interference.

b. Claim 28

Appellants assert that Claim 28 is not estopped on the merits under 35 U.S.C. § 102(g) by the final judgment in the '553 Interference at least because each and every element of Claim 28 was not disclosed or involved in the '553 Interference. During the course of the '553 Interference, the Count was Gluckman Claim 1 from the '460 patent OR Gluckman Claim 1

from the '373 patent OR Lewis Claim 129 from U.S. Patent Application Serial No. 09/064,159 OR Lewis Claim 135 of U.S. Patent Application Serial No. 09/318,001. *Decision on Motion in Interference 104,553, Paper No. 111, ¶¶ 1-5, 12, 13.* During the '553 Interference, the Count was given its broadest reasonable construction. *Decision on Motion in Interference 104,553, Paper No. 111, Page 40.* The Board noted that the "broadest reasonable construction of Gluckman's 460 claim 1 and 373 claim 1 is that the treatment must be for a CNS insult 'affecting' glia or other non-cholinergic cells, but may also affect cholinergic neurons." *Id.* The finding by the Board that Gluckman's claimed method could unintentionally also treat cholinergic cells was the basis for its denial of Gluckman's motion, referenced in the quote above by the Examiner.

In contrast, Appellants' Claim 28 is similar to Claim 16, yet specifies that instead of administering IGF-1, *per se*, an effective amount of a biological analog of IGF-1 is administered, wherein that analog is a naturally-occurring analog, IGF-2, or des 1-3 IGF-1. Like Claim 16, Claim 28 is directed to the treatment of CNS injury wherein the CNS injury *predominantly affects glia.* As noted in the Decision on Motions in the '553 Interference, "neither party discloses a targeted treatment in a patient of one type of CNS cell to the exclusion of other CNS cells." *Decision on Motion in Interference 104,553, Paper No. 111, ¶ 26.* Appellants respectfully submit that the Count from the '553 Interference does not anticipate Claim 28 because it fails to disclose each and every element of Claim 28, *e.g.*, (1) that the CNS injury predominantly affects glia; and (2) that an effective amount of a biological analog of IGF-1 is administered, wherein that analog is a naturally-occurring analog, IGF-2, or des 1-3 IGF-1.

i. Claim 72

Claim 72 depends from Claim 28, but specifies that the CNS injury is a demyelinating disorder. As such, Claim 72 includes all of the limitations of Claim 28, yet adds another limitation. Appellants respectfully submit that the anticipation rejection of Claim 72 under 35 U.S.C. § 102(g) due to purported estoppel on the merits by final judgment in the '553 Interference is in error at least for the same reasons as that rejection is in error for Claim 28. In addition, Claim 72's additional limitation, that the CNS injury is a demyelinating disorder, is absent from the '553 Interference.

ii. Claim 73

Claim 73 depends from Claim 28, but specifies that the CNS injury is multiple sclerosis. As such, Claim 73 includes all of the limitations of Claim 28, yet adds another limitation. Appellants respectfully submit that the anticipation rejection of Claim 73 under 35 U.S.C. § 102(g) due to purported estoppel on the merits by final judgment in the '553 Interference is in error at least for the same reasons as that rejection is in error for Claim 28. In addition, Claim 73's additional limitation, that the CNS injury is multiple sclerosis, is absent from the '553 Interference.

2. Purported Obviousness Under 35 U.S.C. § 103

Obviousness is a question of law, based upon several factual inquiries (known as "the *Graham* factors"), including determining (1) the scope and content of the prior art; (2) the level of ordinary skill in the art; (3) the differences between the claimed invention and the prior art; and (4) whether the differences are such that the claimed invention as a whole would have been obvious to one of ordinary skill in the art at the time the invention was made. *See Ruiz v. A.B.*

Chance Co., 234 F.3d 654, 660 (Fed. Cir. 2000) (citing *Graham v. John Deere Co.*, 383 U.S. 1, 17-18 (1966)).

When making an obviousness rejection, Examiners are instructed to "ensure that the written record includes findings of fact concerning the state of the art and the teachings of the references applied. . . . Factual findings made by Office personnel are the necessary underpinnings to establish obviousness. . . . Office personnel must provide an explanation to support an obviousness rejection under 35 U.S.C. 103. 35 U.S.C. 132 requires that the applicant be notified of the reasons for the rejection of the claim so that he or she can decide how best to proceed. . . . In short, the focus when making a determination of obviousness should be on what a person of ordinary skill in the pertinent art would have known at the time of the invention, and on what such a person would have reasonably expected to have been able to do in view of that knowledge." 72(195) Fed. Reg. 57526, at 57527 (Oct. 10, 2007). Examiners bear the initial burden of factually supporting any *prima facie* conclusion of obviousness and if such a case is not made, "the applicant is under no obligation to submit evidence of nonobviousness." *M.P.E.P. § 2142* (noting also that the "key to supporting any rejection under 35 U.S.C. 103 is the clear articulation of the reason(s) why the claimed invention would have been obvious").

a. Claim 16

Appellants respectfully submit that a *prima facie* case of obviousness as to Claim 16 has not been made. There are no factual findings of record for the '745 application regarding the *Graham* factors. That is, there are no findings regarding: (1) the scope and content of the prior art; (2) the level of ordinary skill in the art; (3) the differences between the claimed invention and the prior art; and (4) whether the differences are such that the claimed invention as a whole would have been obvious to one of ordinary skill in the art at the time the invention was made.

See Ruiz, 234 F.3d at 660. There is no basis, other than the Examiner's own opinion, for the assertion that "the count, which suggests treating glial cells which are injured by multiple sclerosis, also suggests the broader limitation of treating glial cells damaged by CNS injury which predominantly affects glia." *Office Action mailed July 20, 2007, Pages 5-6, ¶ 9.*

Moreover, a party cannot seek to add a claim to an application in interference and request that it be designated as not corresponding to the Count. *L'Esperance v. Nishimoto*, 18 U.S.P.Q.2d 1534 (Bd. Pat. App. & Int. 1991). The Decision on Motions from the '553 Interference notes that because Gluckman could *not* add narrower claims, such as Claim 16, Gluckman would *not be estopped* from filing a reissue application seeking narrower claims that would not have been obvious in view of the subject matter of the lost count to the extent Gluckman's specification supports such claims. *Decision on Motions in Interference 104,553, Paper No. 111, Page 23 n.17.* Claim 16 is narrower than the claims involved in the '553 Interference at least because it specifies that the CNS injury predominantly affects glia. Thus, interference estoppel does *not* attach because Appellants were not able to add such a claim during the course of the '553 Interference.

With regard to the Examiner's citation of 37 C.F.R. § 41.127(a), Appellants submit that this portion of the Code simply states that an interference judgment "disposes of all issues that were, or by motion could have properly been, raised and decided." *37 C.F.R. § 41.127(a).* As explained above and as noted in footnote 17 on Page 23 of the Decision on Motions, Appellants could *not* have presented narrower claims, such as Claim 16, during the interference. Therefore, 37 C.F.R. § 41.127(a) does not apply to Appellants' situation.

With regard to the Examiner's citation of Examples 2 and 3 of M.P.E.P. § 2308.03, Appellants submit that these Examples are similarly inapplicable, but that M.P.E.P. § 2308.03

itself is instructive. The first paragraph of M.P.E.P. § 2308.03 notes that the "time for the party to make all pertinent arguments is during the interference, *unless the Board expressly prevented the party from litigating the issue during the interference.*" Here, the Board expressly prevented Appellants from presenting Claim 16 during the interference. *Decision on Motions in Interference 104,533, Paper No. 111, Page 23, Page 23, n.17.* Yet, the Board also expressly noted in the Decision that Applicants would *not* be estopped from filing a reissue application seeking narrower claims that would not have been obvious in view of the subject matter of the lost Count. *Id.*

With regard to Example 2 in M.P.E.P. § 2308.03, this Example is inapplicable to Appellants' situation because it involves situations where the applicant files a continuation application with a "claim generic to subject matter X [the subject matter of the Count]." Appellants' Claim 16 is not generic to the Count in Interference 104,533, but is instead directed to methods for treating glial cells damaged from CNS injury, wherein said CNS injury *predominantly affects glia* which was neither taught nor claimed by the opponent (Lewis *et al.*) in the '553 Interference.

With regard to Example 3 in M.P.E.P. § 2308.03, this Example is inapplicable to Appellants' situation because it involves situations where the applicant files a continuing application with a "claim to subject matter that would have been obvious in view of subject matter X [the subject matter of the Count]." Appellants' Claim 16 is not obvious in view of the subject matter of the Count in the '553 Interference, but are instead directed to methods for treating glial cells damaged from CNS injury, wherein said CNS injury *predominantly affects glia.* Indeed, Examiner Russel failed to cite any basis other than his own opinion as to why the targeted glial cells would be obvious over a Count construed as treating any and all CNS cells.

i. Claim 66

Claim 66 depends from Claim 16, but specifies that the CNS injury is a demyelinating disorder. As such, Claim 66 includes all of the limitations of Claim 16, and adds another limitation. Appellants respectfully submit that the rejection of Claim 66 under 35 U.S.C. § 103 due to purported estoppel on the merits by final judgment in the '553 Interference is in error at least for the same reasons as that rejection is in error for Claim 16.

Further and as with Claim 16, a *prima facie* case of obviousness has not been made out against Claim 66. There are no factual findings of record for the '745 application regarding the *Graham* factors as they pertain to Claim 66. *See Section VIII.B.2.a, above*. There is no basis, other than the Examiner's own opinion, as to the purported obviousness of Claim 66. *See id.*

Again similar to the situation with Claim 16, Claim 66 is narrower than the claims involved in the '553 interference at least because it specifies that the CNS injury predominantly affects glia, and further specifies that the CNS injury is a demyelinating disorder. Because Appellants could not have added Claim 66 during the interference, interference estoppel does not attach to Claim 66.

ii. Claim 67

Claim 67 depends from Claim 16, but specifies that the CNS injury is multiple sclerosis. As such, Claim 67 includes all of the limitations of Claim 16, and adds another limitation. Appellants respectfully submit that the rejection of Claim 67 under 35 U.S.C. § 103 due to purported estoppel on the merits by final judgment in the '553 Interference is in error at least for the same reasons as that rejection is in error for Claim 16.

Further and as with Claim 16, a *prima facie* case of obviousness has not been made out against Claim 67. There are no factual findings of record for the '745 application regarding the

Graham factors as they pertain to Claim 67. *See Section VIII.B.2.a, above*. There is no basis, other than the Examiner's own opinion, as to the purported obviousness of Claim 67. *See id.*

Again similar to the situation with Claim 16, Claim 67 is narrower than the claims involved in the '553 interference at least because it specifies that the CNS injury predominantly affects glia, and further specifies that the CNS injury is multiple sclerosis. Because Appellants could not have added Claim 67 during the interference, interference estoppel does not attach to Claim 66.

b. Claim 28

Claim 28 is similar to Claim 16, yet specifies that instead of administering IGF-1, *per se*, an effective amount of a biological analog of IGF-1 is administered, wherein that analog is a naturally-occurring analog, IGF-2, or des 1-3 IGF-1. Like Claim 16, Claim 28 is directed to the treatment of CNS injury wherein the CNS injury *predominantly affects glia.* As noted in the Decision on Motions in the '553 Interference, "neither party discloses a targeted treatment in a patient of one type of CNS cell to the exclusion of other CNS cells." *Decision on Motion in Interference 104,553, Paper No. 111, ¶ 26.* Appellants respectfully submit that the Count from the '553 Interference does not render obvious Appellants' Claim 28, which specifically requires that the CNS injury predominantly affects glia.

In addition, Appellants respectfully submit that a *prima facie* case of obviousness has not been made against Claim 28. Obviousness is a question of law, based upon the *Graham* factors, enumerated above. Appellants respectfully submit that a *prima facie* case of obviousness as to Claim 28 has not been made. There are no factual findings of record for the '745 application regarding the *Graham* factors. The record lacks the "[f]actual findings made by Office personnel [that] are the necessary underpinnings to establish obviousness." 72(195) Fed. Reg. 57526, at

57527 (Oct. 10, 2007). There is no basis, other than the Examiner's own opinion, for the assertion that "the count, which suggests treating glial cells which are injured by multiple sclerosis, also suggests the broader limitation of treating glial cells damaged by CNS injury which predominantly affects glia." *Office Action mailed July 20, 2007, Pages 5-6, ¶ 9.*

Moreover, a party cannot seek to add a claim to an application in interference and request that it be designated as not corresponding to the Count. *L'Esperance v. Nishimoto*, 18 U.S.P.Q.2d 1534 (Bd. Pat. App. & Int. 1991). The Decision on Motions from the '553 Interference notes that because Gluckman could *not* add narrower claims, such as Claim 28, Gluckman would *not be estopped* from filing a reissue application seeking narrower claims that would not have been obvious in view of the subject matter of the lost count to the extent Gluckman's specification supports such claims. *Decision on Motions in Interference 104,553, Paper No. 111, Page 23, Page 23, n.17.* Claim 28 is narrower than the claims involved in the '553 Interference at least because it specifies that the CNS injury predominantly affects glia and further specifies that instead of administering IGF-1, *per se*, an effective amount of a biological analog of IGF-1 is administered, wherein that analog is a naturally-occurring analog, IGF-2, or des 1-3 IGF-1. Thus, interference estoppel does *not* attach because Appellants were not able to add such a claim during the course of the '553 Interference.

i. Claim 72

Claim 72 depends from Claim 28, but specifies that the CNS injury is a demyelinating disorder. As such, Claim 72 includes all of the limitations of Claim 28, and adds another limitation. Appellants respectfully submit that the rejection of Claim 72 under 35 U.S.C. § 103 due to purported estoppel on the merits by final judgment in the '553 Interference is in error at least for the same reasons as that rejection is in error for Claim 28.

Further and as with Claim 28, a *prima facie* case of obviousness has not been made out against Claim 72. There are no factual findings of record for the '745 application regarding the *Graham* factors as they pertain to Claim 72. *See Section VIII.B.2.b, above.* There is no basis, other than the Examiner's own opinion, as to the purported obviousness of Claim 72. *See id.*

Again similar to the situation with Claim 28, Claim 72 is narrower than the claims involved in the '553 interference at least because it specifies that the CNS injury predominantly affects glia, and further specifies that the CNS injury is a demyelinating disorder. Because Appellants could not have added Claim 72 during the interference, interference estoppel does not attach to Claim 72.

ii. Claim 73

Claim 73 depends from Claim 28, but specifies that the CNS injury is multiple sclerosis. As such, Claim 73 includes all of the limitations of Claim 28, and adds another limitation. Appellants respectfully submit that the rejection of Claim 73 under 35 U.S.C. § 103 due to purported estoppel on the merits by final judgment in the '553 Interference is in error at least for the same reasons as that rejection is in error for Claim 28.

Further and as with Claim 28, a *prima facie* case of obviousness has not been made out against Claim 73. There are no factual findings of record for the '745 application regarding the *Graham* factors as they pertain to Claim 73. *See Sections VIII.B.2.b, above.* There is no basis, other than the Examiner's own opinion, as to the purported obviousness of Claim 73. *See id.*

Again similar to the situation with Claim 28, Claim 73 is narrower than the claims involved in the '553 interference at least because it specifies that the CNS injury predominantly affects glia, and further specifies that the CNS injury is multiple sclerosis. Because Appellants

could not have added Claim 73 during the interference, interference estoppel does not attach to Claim 73.

3. Conclusion

Appellants respectfully assert that the rejection of Claims 16, 28, 66, 67, 72, and 73 due to purported estoppel on the merits by final judgment in the '553 Interference under 35 U.S.C. §§ 102(g) and/or 103 is improper and should be reversed.

The Examiner has failed to establish how Claims 16, 28, 66, 67, 72, and 73 are anticipated or rendered obvious. Moreover, Claims 16, 28, 66, 67, 72, and 73 are narrower than the claims involved in the '553 Interference and, as such, Appellants could not have added them to the '553 Interference. Therefore, interference estoppel should not attach to Claims 16, 28, 66, 67, 72, and 73.

In view of the foregoing, Appellants respectfully request withdrawal of the rejection under 35 U.S.C. §§ 102(g) and/or 103 of Claims 16, 28, 66, 67, 72, and 73 due to purported estoppel on the merits by final judgment in the '553 Interference.

IX. Claims Appendix

Pursuant to 37 C.F.R. § 41.37(c)(1)(viii), Appellants append hereto, as Appendix 1, a copy of the claims involved in the appeal.

X. Evidence Appendix

Pursuant to 37 C.F.R. § 41.37(c)(1)(ix), Appellants append hereto, as Appendix 2, a copy of the '460 patent.

XI. Related Proceedings Appendix

Because Appellants are aware of a prior interference that is related to and/or directly affects the Board's decision in this Appeal, Appellants, pursuant to 37 C.F.R. § 41.37(c)(1)(x), have appended, in Appendix 3: (1) a copy of the Decision on Motions from the '553 Interference; and (2) a copy of the Judgment from the '553 Interference. *See Section II, supra.*

XII. Conclusion

Appellants respectfully submit that the rejections of Claims 16, 28, 66, 67, 72, and 73 due to (1) estoppel on the merits by final judgment in the '553 Interference under 35 U.S.C. § 103; and (2) estoppel on the merits by final judgment in the '553 Interference under 35 U.S.C. §§ 102(g) and/or 103 should be reversed.

The Director is hereby authorized to charge any additional fees which may be required, or credit any overpayment to Deposit Account No. 50-4047.

Respectfully submitted,
BINGHAM MCCUTCHEN, LLP

Date: March 21, 2008 By: _____
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APPENDIX 1 - CLAIMS APPENDIX

16. A method of treating glial cells damaged from CNS injury, wherein said CNS injury predominantly affects glia, comprising administering to the CNS of a mammal in need thereof, an effective amount of IGF-1.

28. A method of treating glial cells damaged from CNS injury, wherein said CNS injury predominantly affects glia, comprising administering to the CNS of a mammal in need thereof, an effective amount of a biological analog of IGF-1, wherein said analog is selected from the group consisting of naturally-occurring analogs, IGF-2, and des 1-3 IGF-1.

64. A method for treating non-cholinergic cells damaged from CNS injury, comprising administering to the CNS of a mammal in need thereof, an effective amount of IGF-1, wherein the CNS injury is an injury to the hippocampus.

65. A method of treating non-cholinergic cells damaged from CNS injury, comprising administering to the CNS of a mammal in need thereof, an effective amount of a biological analog of IGF-1, wherein the CNS injury is an injury to the hippocampus and further wherein said analog is selected from the group consisting of naturally-occurring analogs, IGF-2, and des 1-3 IGF-1.

66. The method according to Claim 16, wherein said CNS injury is a demyelinating disorder.

67. The method according to Claim 16, wherein said CNS injury is multiple sclerosis.

68. The method according to Claim 16, wherein said CNS injury is periventricular leucomalacia.

69. The method according to Claim 16, wherein said CNS injury is carbon monoxide inhalation.

70. The method according to Claim 16, wherein said CNS injury is ammonia intoxication.

71. The method according to Claim 16, wherein said CNS injury is gaseous intoxication.

72. The method according to Claim 28, wherein said CNS injury is a demyelinating disorder.

73. The method according to Claim 28, wherein said CNS injury is multiple sclerosis.

74. The method according to Claim 28, wherein said CNS injury is periventricular leucomalacia.

75. The method according to Claim 28, wherein said CNS injury is carbon monoxide inhalation.

76. The method according to Claim 28, wherein said CNS injury is ammonia intoxication.

77. The method according to Claim 28, wherein said CNS injury is gaseous intoxication.

APPENDIX 2 - EVIDENCE APPENDIX

Appended hereto is a copy of the '460 patent.

APPENDIX 3 - RELATED PROCEEDINGS APPENDIX

Appended hereto is: (1) a copy of the Decision on Motions from the '553 Interference; and (2) a copy of the Judgment from the '553 Interference.

BOX INTERFERENCE
WASHINGTON DC 20231
703-308-9797
703-305-0942 (fax)

Paper 111

UNITED STATES PATENT AND TRADEMARK OFFICE
BEFORE THE BOARD OF PATENT APPEALS AND INTERFERENCES

PETER GLUCKMAN
and KAROLY NIKOLICS
(5,714,460 and 5,861,373),
Junior Party,

v.

MICHAEL E. LEWIS,
JAMES C. KAUER, KEVIN R. SMITH,
KATHLEEN V. CALLISON, and FRANK BALDINO
(09/064,159 and 09/318,001),
Senior Party.

Interference No. 104,553

Before **TORCZON**, SPIEGEL, and LANE, Administrative Patent Judges.

DECISION ON MOTIONS
(37 C.F.R. § 1.640)

FINDINGS OF FACT

Subject matter of the interference

[1] The subject matter of the interference is the treatment of neural damage or disease using an insulin-like growth factor [IGF]. Count 1, the sole count, is a phantom count that defines the interfering subject matter as the union of a claim from each of the involved patents and applications:

> Gluckman 460 claim 1 OR Gluckman 373 claim 1 OR Lewis 159 claim 129 OR Lewis 001 claim 135.

[2] Gluckman has two involved patents:

P. Gluckman & K. Nikolics, "IGF-1 to improve neural outcome", U.S. Patent 5,714,460 [460] (3 February 1998) [2001][1] and

P. Gluckman & K. Nikolics, "IGF-1 to improve the neural condition", U.S. Patent 5,861,373 [373] (19 January 1999) [2002].

[3] Lewis has two involved applications:

M.E. Lewis et al., "Treating disorders by application of insulin-like growth factors and analogs", patent application 09/064,159 [159] (22 April 1998) [1006] and

M.E. Lewis et al., "Treating disorders by application of insulin-like growth factors and analogs", patent application 09/318,001 [001] (25 May 1999) [1008].

[4] Gluckman 460 claim 1 is [2001 at 13:47-14:3]:

A method of treating neural damage suffered after a CNS insult affecting glia or other non-cholinergic cells in a mammal, comprising administering to the central nervous system of said mammal an effective amount of IGF-1 and/or a biologically active analogue of IGF-1.

[5] Gluckman 373 claim 1 is [2002 at 14:21-26]:

A method of treating neural damage suffered after a CNS insult affecting glia or other non-cholinergic cells in a mammal, comprising administering to the central nervous system of said mammal a medicament comprising an effective amount of IGF-1 and/or a biologically active analog of IGF-1.

[6] "CNS" means central nervous system [2001 at 1:13-14].

[7] The brain and spinal cord constitute the CNS [1031, ¶2].[2]

[8] The nervous system comprises two distinct classes of cells: nerve cells (neurons) and glial cells (glia) [2050 at 19].

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[1] Exhibit numbers starting with "2" are Gluckman exhibits; those starting with "1" are Lewis exhibits.

[2] Paper 38, fact 5, admitted at Paper 58 at 4.

[9] Glia support the activity of neurons, regulate the environment of neurons, and insulate neurons [2024, ¶8; 2001 at 1:40-41; 2050 at 22].

[10] Neurons communicate using small signaling molecules (neurotransmitters). Neurons that use acetylcholine are "cholinergic"; those that do not are "non-cholinergic" [2024, ¶7]. According to Gluckman, non-neuronal cells do not use acetylcholine as a neurotransmitter and are thus non-cholinergic cells (Paper 55 at 8).

[11] "IGF-1" means insulin-like growth factor 1 [2001 at 1:15]. The art appears to use "IGF-I" and "IGF-1" interchangeably [2001 at 2:6-39]. Lewis uses "IGF-I" [1006 at 2:2].[3]

[12] Lewis 159 claim 129 is (Paper 1 at 5 n.1):

> A method of treating a mammal subject to a neurological disease comprising administering to said mammal an amount of insulin-like growth factor I effective to enhance the survival of non-mitotic neuronal cells at risk of dying.

[13] Lewis 001 claim 135 is (Paper 1 at 5 n.1):

> A method of enhancing the survival of CNS neuronal cells in a mammal, said cells being at risk of dying caused by injury or epilepsy, which method comprises administering to said mammal an effective amount of IGF-I.

[14] Gluckman 460 claims 1-15 (all) and Gluckman 373 claim 1 (all) correspond to the count. Lewis 159 claims 129-133 (all) and Lewis 001 claims 135-140 and 142 correspond to the count. Lewis 001 claims 129-134 and 141 do not correspond (Paper 1 at 5-6).

Interference-in-fact

[15] Gluckman contends that there is in fact no interference between the parties (Paper 29). Gluckman contingently contends that if there is an interference-in-fact, then its 460 claims 5-9 do

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[3] This being biotechnology, there are other names as well. For simplicity we use "IGF-1" except in quotes.

not correspond to the count (Paper 34). Lewis contingently moves to add claims 134-136 to its 159 application to ensure an interference exists in fact (Paper 41).

[16] An earlier Lewis patent [2006][4] was cited as a reference in the examination of both involved Gluckman patents [2001 & 2002]. The Gluckman patents were allowed despite the citation of Lewis 317. Gluckman characterized Lewis 317 as describing "[m]ethods for enhancing the survival of cholinergic neuronal cells by administration of IGF-1" [2001 at 2:51-53].

[17] IGF-1 receptors are widespread in the CNS occurring on both glia and neurons [2001 at 2:55-58]. In proposing the interference, the examiner noted (Paper 1, Form PTO-850, Att. D) the wide distribution of IGF-1 receptors throughout adult mammalian brains, including:

> non-mitotic neuronal cells, CNS neuronal cells, glia cells, and non-cholinergic cells[,] all [of which] would have been expected to respond to the administered IGF-I.

[18] Both of the Gluckman claims that are part of the count include a limitation to "treating neural damage suffered after a CNS insult affecting glia or other non-cholinergic cells in a mammal". Gluckman's Field of Invention states [2001 at 1:11-18]:

> This invention relates to methods and therapeutic compositions for the treatment or prevention of central nervous system (CNS) damage and relates particularly although not necessarily to a method of increasing the concentration of insulin-like growth factor 1 (IGF-1) in the central nervous system of the patient to treat an injury or disease that primarily causes damage to glia and/or other non-cholinergic cells of the CNS.

[19] In the Background, Gluckman proceeds to indicate the scope of injury and disease subject to its method [2001 at 1:22-2:5] (citations omitted):

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[4] Michael E. Lewis et al., "Treating disorders by application of insulin-like growth factor", U.S. Patent 5,093,317 [317] (3 March 1992). The inventors on the 317 patent are identical to the present Lewis named inventors.

After asphyxial, traumatic, toxic, infectious, degenerative, metabolic, ischemic or hypoxic insults to the central nervous system (CNS) of man a certain degree of damage in several different cell types may result. For example periventricular leucomalacia, a lesion which affects the periventricular oligodendrocytes is generally considered to be a consequence of hypoxicischemic injury to the developing preterm brain. Further cholinergic neuronal cell bodies are absent from most regions of the cortex in primates and rats. Damage to the cerebral cortex by trauma, asphyxia, ischemia, toxins or infection is frequent and may cause sensory, motor or cognitive deficits. Glial cells which are non-neuronal cells in the CNS are necessary for normal CNS function. Infarcts are a principle component of hypoxicischemic induced injury and loss of glial cells is an essential component of infarction.

Diseases of the CNS also may cause loss of specific populations of cells. For example multiple sclerosis is associated with loss of myelin and oligodendrocytes, similarly Parkinson's disease is associated with loss of dopaminergic neurons. Some situations in which CNS injury or disease can lead to predominant loss of glia or other non-cholinergic cell types or infarction include: perinatal asphyxia associated with fetal distress such as following abruption, cord occlusion or associated with intrauterine growth retardation; perinatal asphyxia associated with failure of adequate resuscitation or respiration; severe CNS insults associated with near miss drowning, near miss cot death, carbon monoxide inhalation, ammonia or other gaseous intoxication, cardiac arrest, collapse, coma, meningitis, hypoglycaemia and status epilepticus; episodes of cerebral asphyxia associated with coronary bypass surgery; cerebral anoxia or ischemia associated with stroke, hypotensive episodes and hypertensive crises; cerebral trauma.

There are many other instances in which CNS injury or disease can cause damage to glia and non-cholinergic neurons of the CNS. It is desirable to treat the injury in these instances. Also, it is desirable to prevent or reduce the amount of CNS damage which may be suffered as a result of induced cerebral asphyxia in situations such as cardiac bypass surgery. To date, there has been no reference in the prior art to the manipulation of insulin-like growth factor 1 (IGF-1) to prevent or treat CNS injury or disease leading to infarction or loss of glia and other non-cholinergic neuronal cells in vivo.

[20] Gluckman explains the role of IGF-1 in neural tissue [2001 at 2:40-3:15] (citations omitted):

IGF-1 is thought to play a paracrine role in the developing and mature brain. In vitro studies indicate that IGF-1 is a potent non-selective trophic agent for several types of neurons in the CNS, including dopaminergic neurons and

oligodendrocytes. Methods for enhancing the survival of cholinergic neuronal cells by administration of IGF-1 have been described.

IGF-1 receptors are wide spread in the CNS occurring on both glia and neurons. These receptors mediate the anabolic and somatogenic effects of IGF-1 and have a higher affinity for IGF-1 compared to insulin. From 3 days after injury greatly increased levels of IGF-1 are produced particularly in the developing CNS. The effect of IGF-1 as a central neuroprotectant when administered after an insult suggests a mode of action involving interference with the activated processes leading to cell death. Endogenous and exogenous IGF-1 stimulate peripheral nerve regeneration. IGF-1 has been shown to enhance ornithine decarboxylase activity in normal rat brains.

It is an object of the invention to provide a method and/or medicament (therapeutic composition) for treating or preventing CNS damage which will go at least some way to meeting the foregoing desiderata in a simple yet effective manner or which will at least provide the public with a useful choice.

[21] Gluckman specifically defines the word "treat" to mean [2001 at 3:25-30]:

> the affecting of a reduction in the severity of the CNS damage, by reducing infarction, and loss of glial cells and non-cholinergic neuronal cells, suffered after a CNS insult. It encompasses the minimising of such damage following a CNS insult.

[22] Gluckman specifically defines the word "prevent" to mean [2001 at 4:9-11]:

> a reduction in the severity of the CNS damage suffered after a CNS insult [and] may also include inhibition of the symptom of CNS damage.

[23] Lewis summarizes its invention in part as follows [1006 at 7:7-8:11]:

> In general, the invention features a method of enhancing the survival of neuronal cells at risk of death, preferably non-mitotic neuronal cells and/or cholinergic neuronal cells, in a mammal, preferably in the context of a therapeutic treatment of neuronal tissues which are suffering from the effects of aging, or injury, or of a disease e.g. Alzheimer's disease, stroke, epilepsy, amyotrophic lateral sclerosis, or Parkinson's disease, by administering to the mammal an effective amount of at least one of the following: IGF-I, a functional derivative of IGF-I, IGF-II, a functional derivative of IGF-II, IGF-III, a functional derivative of IGF-III, with or without the administration of an effective amount of NGF, ciliary neurotrophic factor (CNTF), or a functional derivative thereof.

> The invention also features a method of enhancing the survival of neuronal cells at risk of death, preferably non-mitotic neuronal cells and/or cholinergic neuronal cells, in a mammal, preferably in the context of a therapeutic treatment of neuronal tissues which are suffering from the effects of aging, of injury, or of a disease, e.g., Alzheimer's disease, stroke, epilepsy, amyotrophic lateral sclerosis, or Parkinson's disease, by treating said mammal with a first treatment including administration of a cell survival promoting amount of a growth factor, e.g., IGF-I, IGF-II, or IGF-III, or a functional derivative of the growth factor (e.g., a fragment, analog, or analog of a fragment of the first growth factor), alone, or in a biologically active combination with another such growth factor or functional derivative, and then treating said mammal with a second treatment including administration of a nerve transmitter increasing amount of a transmitter enhancer e.g., NGF, CNTF, or a functional derivative of the transmitter enhancer (e.g., a fragment, analog, or analog of a fragment of the transmitter enhancer). In preferred embodiments, fragments, analogs, or analogs of fragments of IGF-I, IGF-II, IGF-III, or NGF are administered.

[24] Lewis also focuses on treatment of disease and injury [1006 at 9:19-10:5]:

> Another method of the invention features treating a head or spinal cord injury of a mammal, or a disease condition of a mammal, e.g., stroke, epilepsy, age-related neuronal loss, amyotrophic lateral sclerosis, Alzheimer's disease, or Parkinson's disease, by (1), administering to the mammal an effective amount of at least one of the following substances: IGF-, a functional derivative of IGF-I, IGF-II, a functional derivative of IGF-II, IGF-III, a functional derivative of IGF-III, with or without the administration of NGF, CNTF, or a functional derivative thereof, or by (2) treating said mammal with a first treatment including administration of a cell survival promoting amount of one or more of a first group of substances, e.g., IGF-, a functional derivative of IGF-I, IGF-II, a functional derivative of IGF-II, IGF-III, a functional derivative of IGF-III, and then treating said mammal with a second treatment including administration of a nerve transmitter increasing amount of a transmitter enhancer or a functional derivative thereof, e.g., NGF, CNTF, or a functional derivative thereof.

[25] Lewis discloses that enhancing the cholinergic activity of cholinergic neuronal cells is a feature of the invention [1006 at 8:12-9:18], but does not indicate that the scope of disclosure excludes treatment of non-cholinergic cells. Indeed, the background portion of the specification discusses the use of IGF-1 to treat CNS cholinergic and peripheral nervous system [PNS] non-cholinergic

[Schwann] cells [1006 at 5:14-6:18]. The disclosure also notes the benefit of treatment to both neuronal and non-neuronal cells in a PNS injury [1006 at 6:19-22].

[26] As a practical matter, neither party discloses a targeted treatment in a patient of one type of CNS cell to the exclusion of other CNS cells. Gluckman specifically teaches that IGF-1 is "a potent non-selective trophic agent for several types of neurons in the CNS" [2001 at 2:42-44].

Claim correspondence

[27] Gluckman 460 claims 5-9 are [2001 at 14:11-20]:

> 5. A method of claim 1 wherein the central nervous system injury affects non-cholinergic neuronal cells.
>
> 6. A method of claim 1 wherein the central nervous system injury affects glial cells.
>
> 7. A method of claim 1 wherein the central nervous system injury is a consequence of Parkinson's disease.
>
> 8. A method of claim 1 wherein the central nervous system injury is a consequence of multiple sclerosis.
>
> 9. A method of claim 1 wherein the central nervous system injury is a consequence of a demyelinating disorder.

[28] Demyelinating disorders and multiple sclerosis are neurological diseases of the central nervous system (Paper 68 at 9, admitted fact 12).

[29] Dr. Michael Lewis, a Lewis inventor, testified that Parkinson's disease is a neurological disease [1038, ¶8]. According to Gluckman, Parkinson's disease is a disease of dopaminergic neurons [2001 at 1:44-48]:

> Diseases of the CNS also may cause loss of specific populations of cells. For example multiple sclerosis is associated with loss of myelin and oligodendrocytes, similarly Parkinson's disease is associated with loss of dopaminergic neurons.

[30] Lewis identifies Parkinson's disease as a disease subject to treatment with IGF-1 [1006 at 7:7-22]:

> In general, the invention features a method of enhancing the survival of neuronal cells at risk of death, preferably non-mitotic neuronal cells and/or cholinergic neuronal cells, in a mammal, preferably in the context of a therapeutic treatment of neuronal tissues which are suffering from the effects of aging, or injury, or of a disease e.g. Alzheimer's disease, stroke, epilepsy, amyotrophic lateral sclerosis, or Parkinson's disease, by administering to the mammal an effective amount of at least one of the following: IGF-I, a functional derivative of IGF-I, IGF-II, a functional derivative of IGF-II, IGF-III, a functional derivative of IGF-III, with or without the administration of an effective amount of NGF, ciliary neurotrophic factor (CNTF), or a functional derivative thereof.

[31] Gluckman observes that Lewis indicates that Parkinson's disease can be treated by enhancing cholinergic (as opposed to dopaminergic) activity [1006 at 8:12-30].

[32] Dopaminergic neurons and cholinergic neurons are not the same.

[33] Gluckman 460 claims 3, 4, 10, 11, and 14 are [2001 at 13:47-14:42]:

> 3. A method of claim 1[5] wherein the central nervous system injury is ischemic injury.
>
> 4. A method of claim 1 wherein the central nervous system injury is traumatic injury.
>
> 10. A method of claim 1 wherein the IGF-1 and/or biologically active analogue of IGF-1 is administered in the period from the time of the central nervous system injury to 100 hours after the injury.
>
> 11. A method of claim 1 wherein the IGF-1 and/or biologically active analogue of IGF-1 is administered at least once in the period from the time of the central nervous system injury to about 8 hours subsequently.

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[5] 1. A method of treating neural damage suffered after a CNS insult affecting glia or other non-cholinergic cells in a mammal, comprising administering to the central nervous system of said mammal an effective amount of IGF-1 and/or a biologically active analogue of IGF-1.

14. A method of claim 1 wherein the IGF-1 and/or biologically active analogue of IGF-1 is administered to the mammal through a surgically inserted shunt into the cerebro ventricle of the mammal.

[34] Gluckman's disclosure discusses trauma and ischemic injury to the cortex, which Gluckman characterizes as primarily non-cholinergic [2001 at 1:22-40], but 460 claims 3, 4, 10, 11, and 14 are not limited to the cortex.

[35] Cerebral ventricle shunts were established by the mid-1980s as a device for delivering drugs past the blood-brain barrier to treat CNS disease [1028[6] at 171-76].

[36] Lewis 001 claims 129-134 and 141 were not designated as corresponding to the count (Paper 1 at 5-6). The claims are as follows:

129. A method of therapeutically enhancing the survival of CNS neuronal cells in a mammal, said cells being at risk of dying caused by injury or epilepsy, which method comprises administering to said mammal an effective amount of IGF-I or a functional derivative of IGF-I.

130. The method of claim 129 wherein said cells [sic] risk of dying is caused by injury.

131. The method of claim 129 wherein said cells [sic] risk of dying is caused by epilepsy.

132. The method of claim 129 wherein said effective amount of IGF-I or a functional derivative of IGF-I is accompanied by an effective amount of IGF-II or a functional derivative of IGF-II.

133. The method of claim 129 wherein said effective amount is from 0.01 mg/kg to 100 mg/kg of body weight per day.

134. The method of claim 129 wherein said effective amount is administered to the mammal interventricularly into a lateral ventrical.

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[6] A.K. Ommaya, "Implantable devices for chronic access and drug delivery to the central nervous system", 1 Cancer Drug Delivery 169 (1984).

141. A method of therapeutically enhancing the survival of CNS neuronal cells in a mammal, said cells being at risk of dying caused by injury or epilepsy, which method comprises administering to said mammal a pharmaceutical or therapeutic composition comprising an effective amount of IGF-I or a functional derivative of IGF-I

[37] Although the examiner indicated that Lewis 001 claims 129-134 and 141 are not patentable, he also recommended that they correspond to the count [2005]. The declaration (Paper 1) does not explain why this recommendation was not followed.

[38] Gluckman uses Lewis 001 claims 135-140 and 142 as bases for comparison. They are as follows:

135. A method of enhancing the survival of CNS neuronal cells in a mammal, said cells being at risk of dying by injury or epilepsy, which method comprises administering to said mammal an effective amount of IGF-I.

136. The method of claim 135 wherein said cells [sic] risk of dying is caused by injury.

137. The method of claim 135 wherein said cells [sic] risk of dying is caused by epilepsy.

138. The method of claim 135 wherein said effective amount of IGF-I is accompanied by an effective amount of IGF-II.

139. The method of claim 135 wherein said effective amount is from 0.01 mg/kg to 100 mg/kg of body weight per day.

140. The method of claim 135 wherein said effective amount is administered to the mammal interventricularly into a lateral ventrical.

142. A method of therapeutically enhancing the survival of CNS neuronal cells in a mammal, said cells being at risk of dying caused by injury or epilepsy, which method comprises administering to said mammal a pharmaceutical or therapeutic composition comprising an effective amount of IGF-I.

Enablement of the Lewis involved claims

[39] The involved Lewis applications, 159 and 001, have substantially identical disclosures (Lewis Opp. 4, Paper 66, admitted fact 15).

[40] According to Gluckman, there is no distinction between "enhancing survival" and "preventing cell death" as Lewis uses those phrases [2026, ¶3]. The involved Lewis claims require enhancing survival or preventing cell death in non-mitotic or CNS neurons. Mitosis is the process of cell division. The parties agree that CNS neurons are generally not mitotic (Gluckman Reply 4, Paper 58, fact 10).

[41] Lewis did not disclose an example of the *in vivo* use of IGF-1 to treat a CNS injury or disease.

[42] Lewis did provide a number of examples showing IGF-1 effects on CNS tissue [1006 at 44-74]. The examples include *in vivo* administration of IGF-1 to healthy rats [1006 at 49-50].

[43] The *in vivo* examples look for induction of ornithine decarboxylase (ODC) [1006 at 49-50], which was found to increase in response to *in vivo* administration of IGF-1.

[44] The *in vivo* examples included controls [1006, Figs. 5 & 6], which showed lower ODC levels than the test rats. The controls indicate that the procedure itself could not have been solely responsible for the elevated levels of ODC in the test animals.

[45] According to Lewis, ODC induction is a marker for CNS neurotrophic activity [1006 at 49].

[46] According to Gluckman's expert, Dr. David Ginty, "there is evidence to suggest that levels of ODC are not indicative of cell survival." [2040, ¶7]. ODC is not necessary for cell survival [2040, ¶7] and other factors can induce ODC [2040, ¶8].

[47] ODC induction appears to be an early marker for neuron injury and catalyzes polyamine biosynthesis [1012[7] at 191].

[48] Polyamines, in turn, appear to be important to neuronal survival [1013[8] at 178].

[49] Dr. Ginty's challenges to the Lewis disclosure appear to be misdirected toward a search for certainty akin to the sort of results published in a refereed science journal [e.g., 2026, ¶11]:

> Therefore, it is my opinion that none of the Lewis specifications prove that IGF-1 acts to enhance the survival of cholinergic neurons *in vivo*, and certainly do not support claims which encompass enhancing the survival of non-cholinergic neurons or glia.

[50] While many of Dr. Ginty's criticisms appear to be apt, they are not particularly germane to the question of whether any additional experimentation would have been undue [2026 and 2040].

[51] On balance, particularly in view of the *in vivo* controls, ODC appears to provide plausible support for a claim that administration of IGF-1 will promote neuronal survival.

Benefit accorded to Lewis

[52] Lewis was accorded (Paper 1 at 4) the benefit as a constructive reduction to practice of the following U.S. patent applications:

- 08/823,245, filed 24 March 1997 (U.S. Patent 5,776,897);
- 07/958,903, filed 7 October 1992 (U.S. Patent 5,652,214);
- 07/869,913, filed 15 April 1992 (abandoned); and
- 07/534,139, filed 5 June 1990 (abandoned).

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[7] G.M. Gilad & V.H. Gilad, "Polyamine biosynthesis is required for survival of sympathetic neurons after axonal injury", 273 Brain Res. 191 (1983).

[8] G.M. Gilad & V.H. Gilad, "Early polyamine treatment enhances survival of sympathetic neurons after postnatal axonal injury or immunosympathectomy", 38 Dev. Brain Res. 175 (1988).

[53] The Lewis 139 [2028] and 913 [2029] applications disclose the treatment of cholinergic neurons [e.g., 2028 at 7; 2029 at 6-7].

[54] The involved Lewis applications claim the benefit under 35 U.S.C. 120 of Lewis application 07/361,595 [2006],[9] filed 5 June 1989, which issued as the Lewis 317 patent [e.g., 1009 at 2]. The Lewis 139 application is said to be a continuation-in-part of the Lewis 595 application.

[55] The Lewis 317 patent discloses the treatment of injury and disease in cholinergic neurons [2006 at 4:8-22].

[56] The treatment of injury is post-injury [2006 at 4:11-14]:

> preferably in the context of a therapeutic treatment of neuronal tissues which are suffering from the effects of aging, of injury, or of a disease....

[57] Claim 1 of the 317 patent is [2006 at 16:19-22]:

> A method of enhancing the survival of non-mitotic, cholinergic neuronal cells in a mammal, said cells being at risk of dying, said method comprising administering to said mammal an effective amount of IGF-L

The patentability of the Lewis involved claims

[58] Lewis also has an application [2008][10] published under the Patent Cooperation Treaty (PCT) that claims the benefit of the Lewis 595 application.

[59] The Lewis PCT has substantially the same disclosure as the Lewis 595 application.

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[9] The exhibit is the issued patent, not the application. The Federal Circuit has criticized this practice, but also held it to be harmless where there is no material discrepancy. In re Huston, 308 F.3d 1267, 1270 n.1, 64 USPQ2d 1801, 1802 n.1 (Fed. Cir. 2002). There was no timely objection to this exhibit.

[10] M.E. Lewis et al., "Treating disorders by application of insulin-like growth factors and analogs", WO 90/14838, published 13 December 1990 [Lewis PCT]. The inventors are the same as the present Lewis named inventors.

[60] The Lewis PCT discloses in a single reference all of the claimed limitations of the Lewis claims constituting the count.

[61] The publication date for the Lewis PCT application, 13 December 1990 is more than one year prior to the filing date of the Lewis 913 application (14 April 1992), but is not prior to the filing dates of the Lewis 139 application (5 June 1990) and the Lewis 595 application (5 June 1989).

Benefit accorded to Gluckman

[62] Gluckman 460 issued from U.S. patent application 08/460,365, filed 2 June 1995.

[63] Gluckman 373 issued from U.S. patent application 08/500,273, filed 10 July 1995.

[64] Gluckman was accorded (Paper 1 at 3) the benefit as a constructive reduction to practice of:

- 08/185,804, filed 28 January 1994 (abandoned) and
- PCT/US92/06389, filed 3 August 1992.

[65] Gluckman claims benefit via 35 U.S.C. 119 of its 1991 New Zealand provisional specification.[11]

[66] Lewis 159 claim 129 is one alternative of the count, which is:

> A method of treating a mammal subject to a neurological disease comprising administering to said mammal an amount of insulin-like growth factor I effective to enhance the survival of non-mitotic neuronal cells at risk of dying.

[67] The New Zealand provisional application provides the following relevant disclosure [1020 at 2]:

> This invention relates to a method and/or medicament for the treatment or prevention of neural damage and relates particularly although not necessarily solely to a method of increasing the concentration of IGF-I in the brain of the patient to treat neural damage or reducing the concentration of IGF-I in the brain of the patient prior to a potential neural insult so as to reduce the degree of neural damage suffered.

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[11] "Improvements in or relating to a method of treating", New Zealand Prov. Spec. 239211 (1 August 1991). One of the Gluckman inventors, Karoly Nikolics, is a resident of the United States. It is not clear on this record whether Gluckman complied with 35 U.S.C. 184[1]. Gluckman has since received authorizations for foreign filing so it would appear that a retroactive license, if needed, would be readily granted. 37 C.F.R. § 5.25.

[68] The New Zealand provisional application further lists a variety of injuries and diseases that may be so treated [1020 at 2-3].

[69] Direct administration of IGF-1 to a patient is a preferred embodiment [1020 at 4:4-5].

[70] Gluckman asphyxiated test and control rats, treated the test rats with recombinant human IGF-1, and then examined the test rats against control rats. Therapy with IGF-1 was reported to have reduced neuronal damage [1020 at 17-19].

[71] Gluckman reports very favorable results, but the data listed for both experiment A and experiment B show greater neuronal loss for test animals compared with the controls [1020 at 19-21].

[72] Gluckman contends that the data in the table was reversed. Lewis suggests that the data is erroneous. The simplest way to reconcile the bad results with the good discussion is to accept that the data is reversed because many more changes would have to be assumed to bring the discussion in line with the data.

Patentability of the Gluckman claims in view of prior art

[73] The Mozell article[12] is prior art to Gluckman.

[74] The McMorris article[13] is prior art to Gluckman.

[75] The Lewis PCT published application is prior art to Gluckman.

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[12] R.L. Mozell and F.A. McMorris, "Insulin-like growth factor I stimulates regeneration of oligodendrocytes *in vitro*", 540 Annals N.Y Acad. Sci. 430 (1988) [1019]. Page 431 is missing from 1019.

[13] F.A. McMorris, R.W. Furlanetto, R.L. Mozell, M.J. Carson, and D.W. Raibel, "Regulation of oligodendrocyte development by insulin-like growth factors and cyclic nucleotides", 605 Annals N.Y Acad. Sci. 101 (1990) [1018]. It is not clear from the record when in 1990 the McMorris paper was published or whether it was available earlier, for instance as a paper presented at a conference.

[76] If Gluckman is entitled, under 35 U.S.C. 119 and 120, to the benefit of its New Zealand provisional application (August 1991), then Mozell (1988) is available as a reference under 35 U.S.C. 102(b) because it was published more than one year earlier, but the Lewis PCT (December 1990) is only available under 35 U.S.C. 102(a), while the availability of McMorris (1990) under § 102(b) has not been established, but it is available under § 102(a). If Gluckman is not entitled to the benefit of its New Zealand provisional application, then all three references are available under §102(b).

[77] The New Zealand provisional application discloses treating neural damage suffered after a CNS insult [1020 at 17-22].

[78] The disclosed injury included injury to the hippocampus, dentate gyrus, and lateral cortex [1020 at 17].

[79] The New Zealand provisional application does not discuss the treatment of glia or non-cholinergic cells specifically, nor does it teach not treating cholinergic neurons.

[80] We find credible the testimony of Dr. Ginty (2026, ¶13) that the hippocampus contains non-cholinergic neurons (but not the implication that it contains few, if any, cholinergic neurons).

[81] We find credible the testimony of Dr. Ginty (2026, ¶13) that the dentate gyrus contains non-cholinergic neurons (but not the implication that it contains few, if any, cholinergic neurons).

[82] We find credible the testimony of Dr. Ginty (2026, ¶13) that the lateral cortex contains non-cholinergic neurons (but not the implication that it contains few, if any, cholinergic neurons).

[83] The disclosed treatment [1020 at 17] involves an injury "affecting" non-cholinergic neurons.

[84] The New Zealand provisional application does not disclose treatment of neural damage where the injury is from Parkinson's disease, multiple sclerosis, or a demyelinating disorder.

[85] The New Zealand provisional application discloses treatment of asphyxiated rats two hours after termination of the asphyxiation [1020 at 18:7-10]. Gluckman has not pointed out a disclosure of treatment of up to about 8 or 100 hours.

[86] The Lewis PCT published application teaches [1017 at 8:26-9:8]:

> Another method of the invention features treating a head or spinal cord injury of a mammal, or a disease condition of a mammal, e.g., stroke, epilepsy, age-related neuronal loss, amyotrophic lateral sclerosis, Alzheimer's [disease], or Parkinson's [disease], by (1), administering to the mammal an effective amount of at least one of the following substances: IGF-I, a functional derivative of IGF-I, IGF-II, or a functional derivative of IGF-II, with or without the administration of NGF or a functional derivative thereof, or by (2) []treating said mammal with a first treatment including administration of a cell survival promoting amount of one or more of a first group of substances, e.g., IGF-, a functional derivative of IGF-I, IGF-II, or a functional derivative of IGF-II, and then treating said mammal with a second treatment including administration of a nerve transmitter increasing amount of a transmitter enhancer or a functional derivative thereof, e.g., NGF or a functional derivative thereof.

[87] The portion cited teaches the treatment of neural damage suffered after a CNS insult ("treating a head or spinal cord injury of a mammal, or a disease condition of a mammal") comprising administering IGF-1 ("administering to the mammal an effective amount of at least one of the following substances: IGF-I...").

[88] At least one disease[14] so treated, Parkinson's disease, affects non-cholinergic (in this case dopaminergic) neurons.

[89] Assuming, as Gluckman contends, that Lewis thought Parkinson's disease involved injury to cholinergic neurons [see 1017 at 7:22-29], there is no reason in this record to suppose a person having ordinary skill in the art would have made the same mistake.

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[14] Other listed injuries and diseases, including stroke and amyotrophic lateral sclerosis affect a range of tissue. The damage of a stroke is not specific to one kind of neuron as opposed to another. In ALS, collateral damage affects CNS myelin.

[90] The Lewis PCT published application further teaches [1017 at 20:29-36]:

> Thus, the peptides of this invention should be useful for administration to humans or other mammals who suffer from neurological diseases or disturbances characterized by increased risk of neuronal cell death, as described above. These neurological diseases or disturbances include but are not limited to: Alzheimer's disease, Parkinson's disease, amyotrophic lateral sclerosis, stroke, and concussive or penetrating injuries of the brain or spinal cord.

[91] The McMorris article teaches [1018 at 101] that understanding the regulation of oligodendrocyte development and myelin synthesis in the CNS "may ultimately lead towards the development of treatments to promote remyelination in multiple sclerosis and other demyelinating diseases." McMorris concludes [1018 at 105] that IGF-1 "induces the synthesis and accumulation of myelin as well as the development of oligodendrocytes".

[92] Multiple sclerosis is not one of the diseases that the cited portions of the Lewis PCT published application lists.

[93] The Mozell article teaches [1019 at 430] that:

> Because multiple sclerosis is an episodic disease whose primary target is myelin, it was of interest to investigate if IGF-I could stimulate oligodendrocyte development and remyelination after a demyelinating episode.

[94] Mozell concluded [1019 at 432] that "[r]esults using this technique clearly show that IGF-I plays an important role in the regeneration of oligodendrocytes after demyelination."

DISCUSSION

I. Interference in fact

Gluckman preliminary motion 1 (Paper 29) seeks a judgment of no interference-in-fact. Claims that are patentably distinct do not interfere. Nitz v. Ehrenreich, 537 F.2d 539, 544-45, 190 USPQ 413, 417-18 (CCPA 1976). Patentable distinction is determined using the standard

tools of anticipation and obviousness applied to the respective claims of the parties. 37 C.F.R. §1.601(j) & (n). Gluckman specifies two bases of patentable distinction. First, Gluckman contends its claims are limited to glial or non-cholinergic cells, while Lewis's claims are limited to either cholinergic cells or neurons generally. Second, Gluckman contends that its claims are limited to treatment after a CNS insult, while Lewis's are not. According to Gluckman, an obviousness or anticipation analysis would not overcome these distinctions.

The first step in any patentability analysis is a determination of the scope and meaning of the claims with particular focus on the contested limitations. Gechter v. Davidson, 116 F.3d 1454, 1457, 43 USPQ2d 1030, 1032 (Fed. Cir. 1997). The starting place for analysis is the language of the claim because of the heavy presumption that a contested term has the meaning those skilled in the relevant art would ordinarily ascribe to it. Texas Digital Sys. v. Telegenix, Inc., 308 F.3d 1193, 1201-02, 64 USPQ2d 1812, 1817 (Fed. Cir. 2002). In proceedings before the United States Patent and Trademark Office, claims are construed as broadly as reasonably possible in view of the associated specification. In re Sneed, 710 F.2d 1544, 1548, 218 USPQ 385, 388 (Fed. Cir. 1983). In particular, claims should not be limited to examples and embodiments in the specification unless one skilled in the art would read the claims to be so limited; to proceed otherwise would be to read limitations impermissibly from the specification into the claims. Texas Digital, 308 F.3d at 1204, 64 USPQ2d at 1820.

Each party has two claims that are constituents of the count. Both Gluckman claims require "treating neural damage suffered after a CNS insult affecting glia or other non-cholinergic cells in a mammal". Lewis 159 claim 129 requires "treating a mammal subject to a neurological disease...to enhance the survival of non-mitotic neuronal cells at risk of dying", while Lewis 001

claim 135 requires "enhancing the survival of CNS neuronal cells in a mammal, said cells being at risk of dying caused by injury or epilepsy". Both parties treat mammals with neurological conditions. Lewis 159 claim 129 specifies "a neurological disease", while the Gluckman claims specify "neural damage suffered after a CNS insult affecting glia and other non-cholinergic cells". The Gluckman disclosure provides a list of diseases as well as other situations that involve a predominant loss of glia and other non-cholinergic cells. The Gluckman claims making up the count require an "insult affecting glia or non-cholinergic cells", but are not limited to conditions in which glia and other non-cholinergic cell loss predominates.[15]

Lewis, on the other hand, while concentrating on examples showing cholinergic neurons, also discloses IGF-1 treatment of other cell types, including glia and other non-cholinergic cells. A consideration of each Lewis application as a whole fails to show any indication that Lewis intended to be limited strictly to cholinergic neurons. The examiner in proposing the interference quite credibly found that IGF-1 receptors are widely distributed throughout the CNS and would have been expected to have similar beneficial effects on all of the cell types under discussion. The CNS involves a variety of interrelated and interdependent cells. Glia in particular play a critical supporting role for neurons. Treatment of CNS diseases with IGF-1 will result in the non-selective treatment of all exposed CNS cells that respond to IGF-1. Treatment of glia will necessarily enhance the survival of neurons dependent on glia. Conversely, treatment of

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[15] Gluckman's reliance on prosecution history estoppel to limit its claims is misplaced, as is its reliance on any purported inconsistency between the examiners of its applications and the examiner who proposed the interference. The simple answer is that neither we nor Lewis are bound by the ex parte analyses of the examiners in Gluckman's prosecutions. Glaxo Wellcome, Inc. v. Cabilly, 56 USPQ2d 1983, 1984 (BPAI 2000) (interference panel and other party not bound by prior ex parte determinations), citing Switzer v. Sockman, 333 F.2d 935, 942, 142 USPQ 226, 232 (CCPA 1964) and Sze v. Bloch, 458 F.2d 137, 141, 173 USPQ 498, 501(CCPA 1972). Note that if ex parte determinations were binding, Gluckman would not be able to move for no interference-in-fact.

cholinergic neurons will enhance the survival of the glia that exist to serve those neurons. The distinctions Gluckman would have us draw are not required by the language of the claims and are too fine to reflect the uses disclosed.

Another way of understanding the problem is by analogy to "range" cases like In re Woodruff, 919 F.2d 1575, 16 USPQ2d 1934, 1936-37 (Fed. Cir. 1990) (shifting the burden of going forward to the applicant when the claimed range overlaps the prior art range). Lewis has disclosed that IGF-1 treats neurological diseases and tissue generally. Gluckman is using the same compound in substantially the same way to treat particular cells within the neurological tissues that Lewis treats. On its face, Gluckman is doing precisely what a person having ordinary skill in the art would have expected from the Lewis disclosure. Gluckman has not explained why a person having ordinary skill in the art would have expected that the Lewis method would only work within the CNS on cholinergic neurons.

Gluckman also distinguishes between pre-injury and post-injury treatment. Again, the distinction proposed is too fine. According to Gluckman, its claims address post-injury uses ("treating neural damage after a CNS insult"), while the Lewis claims address pre-injury uses ("to enhance the survival of non-mitotic neuronal cells at risk of dying" and "enhancing survival of CNS neuronal cells...at risk of dying caused by injury or epilepsy"). Gluckman's claims are directed to post-injury treatment. The broadest reasonable construction of the plain language of the Lewis claims, however, does not exclude post-injury treatment and even suggests it ("risk of dying caused by injury or epilepsy"). At most, the Lewis claims are directed to two species, pre-injury treatment and post-injury treatment, without specifying a preference between the two. The Lewis specification strongly suggests post-injury treatment is the better reading ("treating a head

or spinal cord injury").[16] Gluckman suggests that Lewis is not entitled to such claims, but that is not a proper issue in the context of a no interference-in-fact motion. If the Lewis claims are unsupported, the remedy is to file a motion attacking the support for those claims, which can properly be done in the context of an interference. 37 C.F.R. §1.633(a). A lack of entitlement to any interfering claim can be a reason to terminate an interference, but it is distinct from the question of whether an interference exists in the first instance.

The patent applicant (in this case, now patentee) is responsible for defining what it regards as its invention. In re Morris, 127 F.3d 1048, 1056, 44 USPQ2d 1023, 1029 (Fed. Cir. 1997). Defining the invention broadly carries the risk that it will interfere with what another has invented, even when the specifics of the inventions differ. Woods v. Tsuchiya, 754 F.2d 1571, 1578 n.5, 225 USPQ 11, 15 n.5 (Fed. Cir. 1985). If the applicant or patentee does not intend to claim so broadly or does not believe it has claimed so broadly, it must distinguish its claims or amend them. Morris, 127 F.3d at 1057, 44 USPQ2d at 1030. In this case, Gluckman has endeavored to distinguish its claims.[17] The distinction, however, requires an interpolation of limitations from the specification to narrow Gluckman's claims.

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[16] [1006 at 9:19-20].

[17] The interference rules do not provide for a movant under 37 C.F.R. §1.633(b) to amend its claims to avoid an interference because the agency would be unable to determine with finality whether the non-movant is entitled priority. Cf. 37 C.F.R. §1.662(b) (treating the filing of a reissue application without corresponding claims as a concession of priority). Since Gluckman cannot so move, if Gluckman were to lose on priority, it would not be estopped (37 C.F.R. §1.658(c)) from filing a reissue application seeking narrower claims that would not have been obvious in view of the subject matter of the lost count to the extent Gluckman's specification supports such claims. E.g., In re Johnson, 558 F.2d 1008, 1018, 194 USPQ 187, 196 (CCPA 1977) (Claims amended in continuing application to exclude subject matter of lost count).

Upon consideration of Gluckman preliminary motion 1, the Lewis opposition, the Gluckman reply, and the portions of the exhibits the parties cited and discussed, we[18] remain of the opinion that the involved claims of the parties interfere. 35 U.S.C. 135(a). Consequently, Gluckman preliminary motion 1 is DENIED.

Lewis preliminary motion 3 is contingent on the granting of Gluckman preliminary motion 1 (Paper 41). That contingency having not been met, Lewis preliminary motion 3 is DISMISSED as moot. Consequently, the Gluckman miscellaneous motion (Paper 82) to strike Lewis reply 3 is also DISMISSED as moot.

Since Gluckman has not received relief as a result of its preliminary motion 1, the Lewis miscellaneous motion to suppress Gluckman reply 1 and related evidence is DISMISSED as moot.

II. Designation of Gluckman 460 claims as not corresponding to the count

A. Gluckman 460 claims 5-9

In Gluckman preliminary motion 6 (Paper 34), Gluckman moves, contingent on the denial of its no interference-in-fact motion, to have its 460 claims 5-9 designated as not corresponding to the count. The contingency has been met, so we address the correspondence of the claims. A claim corresponds to the count if the party losing on priority would not be entitled to the subject matter of the claim. See In re Deckler, 977 F.2d 1449, 1452, 24 USPQ2d 1448, 1449 (Fed. Cir. 1992) (losing party not entitled to claims patentably indistinct from lost count).[19]

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[18] On the Director's behalf. See 37 C.F.R. §1.610(a) & (b) (delegating power to decide such motions).

[19] Lewis' reliance on the two-way test of Winter v. Fujita, 53 USPQ2d 1234 (BPAI 1999), is misplaced. Winter was concerned with the question of whether an interference existed. The question here is whether, assuming Gluckman loses the priority determination for the present count, it should also lose any entitlement to the corresponding

Claims 5 and 6 are not patentably distinct from the subject matter of the count. The count includes Gluckman 460 claim 1, which is in a sense a Markush claim requiring an effect on glia or other non-cholinergic cells. Claim 5 further limits Gluckman's invention to injuries affecting non-cholinergic cells, while claim 6 further limits the invention to injuries affecting glia. Claim 1 expressly teaches both of these alternatives and hence anticipates both claims.[20]

The following table provides the further limitations and arguments for the separate patentability of Gluckman's claims 7, 8, and 9:

Claim	"A method of claim 1 wherein the central nervous system injury is a consequence of ____."	Argument (Paper 34 at 15)
7	"Parkinson's disease"	"a disorder primarily affecting dopaminergic [non-cholinergic] cells"
8	"multiple sclerosis"	"a disorder primarily affecting glia"
9	"a demyelinating disorder"	"disorders affecting glia"

The argument points out differences between the subject matter of the count and the specific claims, but those differences involve the selection of a specific disorder or set of disorders within the scope of the count. Neither Gluckman nor Lewis purport to have been the first to identify these disorders as involving neurons or glia. A person having ordinary skill in the art would have immediately apprehended specific diseases within the set of known CNS diseases that are the subject matter of the count.

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claims. Following Deckler, this question is resolved using a one-way test starting with the subject matter of the lost count.

[20] Indeed, since Gluckman defines glia as non-cholinergic cells, the glia alternative in claim 1 anticipates both claim 5 and claim 6.

Gluckman notes that Lewis appears to link treatment of Parkinson's disease with an increase in cholinergic activity. According to Gluckman, this means that Lewis did not possess the subject matter of Gluckman 460 claims 7-9. The argument fails for three reasons. First, the involved Lewis applications are not prior art for the purpose of determining whether the claims in question would have been obvious to a person having ordinary skill in the art from the subject matter of the lost count. Consequently, whether Lewis meant to imply that Parkinson's disease is a disease of cholinergic neurons and, if so, whether that implication is correct, is entirely irrelevant to the present question of whether Gluckman's claims would have been obvious to a person having ordinary skill in the art in view of the lost count. Second, assuming it is relevant and wrong, Lewis's disclosure of treating Parkinson's disease with IGF-1 is still supported because Lewis did not have to understand why his invention worked. Newman v. Quigg, 877 F.2d 1575, 1581, 11 USPQ2d 1340, 1345 (Fed. Cir. 1989). Finally, the fact that Parkinson's disease involves the impairment of dopaminergic neurons does not by itself mean that increasing cholinergic activity would not have a beneficial effect. Lewis's specification asserts that it does; Gluckman provides no basis for believing that it does not. Given the interrelatedness of neurological cells in the CNS, a relationship is by no means implausible. Since Gluckman, as the movant, has the burden of going forward, the failure to adduce positive proofs on this point works against Gluckman.

Upon consideration of Gluckman preliminary motion 6, the Lewis opposition, the Gluckman reply, and the portions of the exhibits the parties cited and discussed, we remain of the opinion that Gluckman 460 claims 5-9 correspond to the count. Consequently, Gluckman preliminary motion 6 is DENIED.

Since Gluckman has not received relief as a result of its preliminary motion 6, the Lewis miscellaneous motion to suppress Gluckman reply 6 and related evidence is DISMISSED as moot.

B. Gluckman 460 claims 3, 4, 10, 11, and 14

In Gluckman preliminary motion 8 (Paper 36), Gluckman moves to have Gluckman 460 claims 3, 4, 10, 11, and 14 designated as not corresponding to the count. The claims in question fall into two groups. The first group, as with claims 5-9, identify specific kinds of injury:

Claim	"A method of claim 1 wherein the central nervous system injury is ___."	Argument (Paper 36 at 14)
3	"ischemic injury"	"directed to treatment after ischemic injury"
4	"traumatic injury"	"directed to treatment after traumatic injury"

while the second group identify additional limitations to the method itself:

Claim	"A method of claim 1 wherein the IGF-1 and/or biologically active analogue of IGF-1 is administered ___."	Argument (Paper 36 at 14)
10	"in the period from the time of the central nervous system injury to 100 hours after the injury"	"directed to treatment performed within 100 hours after injury"
11	"at least once in the period from the time of the central nervous system injury to about 8 hours subsequently"	"directed to treatment performed within 8 hours after injury
14	"to the mammal through a surgically inserted shunt into the cerebro ventricle of the mammal"	"directed to treatment by surgically inserting a shunt into the cerebro ventricle of the mammal"

Although Gluckman points out additional limitations in these claims, Gluckman does not explain why a person having ordinary skill in the art would have concluded that these limitations were not obvious. In the absence of a specific argument, we are at a loss to know whether to

infer that Gluckman (1) knows of no reason why they would be obvious, (2) is relying on the timing of treatment as the basis for patentable distinctness, or (3) is simply preserving its options. We note that claims 3 and 4 are directed to the treatment of specific CNS injuries with a method that the record suggests has general CNS applicability. Absent some explanation, it is difficult to see why these claims are directed to an invention different from the subject matter of the count. Claims 10 and 11 cover any prompt use of the method. All other considerations being equal, prompt treatment is generally indicated for serious injuries, particularly to the CNS. Again, without some explanation, it is difficult to imagine such claims being separately patentable. Claim 14 further limits the method of administration to the use of a shunt surgically inserted into the cerebro ventricle. Given the problems of protecting polypeptides from degradation during other modes of administration and of passing large molecules through the blood-brain barrier, direct administration of a polypeptide like IGF-1 to the CNS using established methods including injection, which both parties disclose, and a shunt as Ommaya teaches would seem, absent some explanation, to be routine. A motion to have claims designated as not corresponding to the count places the movant in the awkward position of having to prove a negative. Consequently, the burden of making out a case is fairly low, but it is not zero. Were these the only reasons advanced, the motion would be denied for failure to make out a prima facie case. Gluckman's motion, however, explicitly relies on a more general reason why these claims are patentably distinct.

According to Gluckman, its claims are directed to treatment after a CNS insult, while the Lewis claims are not (Paper 36 at 14-15). The problem is that Gluckman's analysis turns on the purported failure of Lewis to enable post-injury treatment. This analysis mixes remedies. If the

Lewis claims are not enabled, the proper remedy is a motion under §1.633(a) for a judgment of unpatentability against the Lewis involved claims. Gluckman has, in fact, filed such a motion (Paper 32, discussed below). For the purpose of determining correspondence (as in the case of interference-in-fact), the claims are taken at their face value.

Gluckman also argues that Lewis conceded the separate patentability of its claims to enhancing survival and its claims to treating neural damage when it failed to traverse a restriction requirement between such claims (Paper 36 at 17). As concessions go, the failure to traverse a restriction, while procedurally binding, has little probative value. Restrictions are made for administrative reasons. In re Weber, 580 F.2d 455, 458, 198 USPQ 328, 332 (CCPA 1978). It follows that an applicant might accede to such a requirement out of administrative convenience rather than as an affirmative concession on the merits of the requirement. Cf. Bayer Aktiengesellschaft v. Duphar Int'l Research B.V., 738 F.2d 1237, 1243, 222 USPQ 649, 653 (Fed. Cir. 1984) (noting little connection between electing under a restriction requirement and surrendering scope under the doctrine of equivalents).

Upon consideration of Gluckman preliminary motion 8, the Lewis opposition, the Gluckman reply, and the portions of the exhibits the parties cited and discussed, we remain of the opinion that Gluckman 460 claims 3, 4, 10, 11, and 14 correspond to the count. Consequently, Gluckman preliminary motion 8 is DENIED.

Since Gluckman has not received relief as a result of its preliminary motion 8, the Lewis miscellaneous motion to suppress Gluckman reply 8 and related evidence is DISMISSED as moot.

III. Designation of additional Lewis claims as corresponding

Gluckman preliminary motion 5 (Paper 33) seeks a designation of Lewis 001 claims 129-134 and 141 as corresponding to the count. These claims currently stand rejected.

Comparison of Lewis 001 claims 129-134 and 141 with Lewis 001 claims 135-140 and 142 reveals two differences. The first, the addition of the word "therapeutically" before "enhancing the survival" in claim 129 (compared to claim 135)[21] appears to be an immediately obvious application of enhancing CNS neuronal cell survival in mammals. The second difference, and the one on which Lewis relies, is the inclusion in claims 129 and 141 of "or a functional derivative of IGF-I". This additional alternative does not, however, render the first alternative, using IGF-I itself, separately patentable from the subject matter of claims 135 and 142, respectively, which use only IGF-I. Phrased differently, if we imagine that the subject matter of claims 135 and 142 was known in the prior art, then the subject matter of claims 129 and 141 would be anticipated despite the addition of a (possibly) patentably distinct alternative of using a derivative of IGF-I.

As noted previously, correspondence is a prediction of what the examiner would be able to reject over a lost count. The examiner has already indicated an intent to reject claims 129-134 and 141 in the event Lewis loses the interference. That intent appears credible in view of nearly identical language of the claims and the unconvincing arguments of Lewis. Upon consideration

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[21] The Gluckman motion is a bit confusing in that it attempts to compare claim 129 with claim 138, which adds a further limitation to claim 135. The comparison with claim 135 is more natural as Lewis appears to acknowledge by pointing out the irrelevance of the additional limitation from claim 138. Although Lewis is correct that Gluckman's comparison is inapposite, the correct comparison is readily apparent. Given the examiner's intent to reject these claims as if they corresponded to the count anyway, reliance on a technicality to deny the motion would not increase the speed, cost-efficiency, or fairness of the interference.

of Gluckman preliminary motion 5 and the Lewis opposition, we are of the opinion that Lewis 001 claims 129-134 and 141 correspond to the count. Consequently, Gluckman preliminary motion 5 is GRANTED.

IV. Compliance of Lewis involved claims with the written description and enablement requirements of 35 U.S.C. 112[1]

Gluckman preliminary motion 4 (Paper 32) seeks a judgment of unpatentability against the involved claims in the Lewis applications on the grounds that the claims lack a written and enabling description. According to Gluckman (Paper 32 at 2), Lewis does not enable enhanced survival of neuronal cells *in vivo*.

In interferences, a patentability analysis must focus on the patentability of individual claims. In re Van Geuns, 988 F.2d 1181, 1186, 26 USPQ2d 1057, 1060 (Fed. Cir. 1993). Neither the motion nor the opposition, however, singles out any of the Lewis claims for separate treatment. Rather, the motion focuses on two broad attributes of the Lewis claims that are said to lack enabling support: the type of cells treated (neurons generally or cholinergic cells specifically) and the purpose of the method (enhancing cell survival or preventing cell death). In deciding this motion, we treat the Lewis claims as standing or falling together based on the support provided in the respective involved Lewis applications. In re Brana, 51 F.3d 1560, 1566 n.19, 34 USPQ2d 1436, 1441 n.19 (Fed. Cir. 1995) (enablement must exist as of the filing date of the application containing the claims in question). The question of benefit must be addressed

separately.[22] Cf. Reiffin v. Microsoft Corp., 214 F.3d 1342, 1346, 54 USPQ2d 1915, 1918 (Fed. Cir. 2000) (distinguishing between §112[1] support and §120 benefit).

A conclusion of non-enablement under §112[1] is appropriate where the written description fails to teach those in the art to make and use the invention as broadly as it is claimed without undue experimentation. In re Cortright, 165 F.3d 1353, 1356, 49 USPQ2d 1464, 1466 (Fed. Cir. 1999). According to Gluckman (Paper 32 at 19), Lewis failed to disclose *in vivo* models for the *in vivo* activity of its claims. Gluckman provides expert testimony that without *in vivo* results, artisans of ordinary skill would not have a reasonable expectation of success.

There is no per se rule requiring *in vivo* testing to enable claims to *in vivo* activity.[23] Rather the evidence in the disclosure must reasonably correlate to the claimed activity. Fujikawa v. Wattanasin, 93 F.3d 1559, 1565, 39 USPQ2d 1895, 1900 (Fed. Cir. 1996). Elevated ODC induction correlates to neuronal survival. Gluckman relies heavily on its expert, Dr. Ginty, to challenge the Lewis disclosure. Dr. Ginty points to many weaknesses in the Lewis disclosure but does not explain how those weaknesses would have prevented one skilled in the art from practicing the claimed method. Significantly, Dr. Ginty does not state that one skilled in the art would not have been able to make the method work. Rather he says that Lewis did not prove it would. Scientific certainty is not the standard for an enabling disclosure.

Upon consideration of Gluckman preliminary motion 4, the Lewis opposition, the Gluckman reply, and the portions of the exhibits the parties cited and discussed, we cannot hold

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[22] Gluckman has filed a motion under 37 C.F.R. §1.633(g) (Paper 35) attacking accordance of benefit to Lewis for two of its earlier applications as constructive reductions to practice.

[23] Note that as movant, Gluckman has the burden of proof.

the Lewis claims unpatentable for lack of enabling support. Consequently, Gluckman preliminary motion 4 is DENIED.

Since Gluckman has not received relief as a result of its preliminary motion 4, the Lewis miscellaneous motion to suppress Gluckman reply 4 and related evidence is DISMISSED as moot.

V. Entitlement of Lewis to the benefit of its 913 and 139 applications

Gluckman preliminary motion 7 (Paper 35) attacks the benefit accorded to Lewis for its constructive reduction to practice of an embodiment within the scope of the count. Specifically, Gluckman argues that the Lewis 913 and 139 applications do not provide enabling support for its claims in interference (Paper 35 at 1).

Gluckman filed its motion under 37 C.F.R. §1.633(g), which authorizes the movant:

> to attack the benefit accorded an opponent in the notice declaring the interference of the filing date of an earlier filed application. See §1.637 (a) and (g).

In turn, 37 C.F.R. §1.637(g) requires:

> A preliminary motion to attack benefit under § 1.633(g) shall explain, as to each count, why an opponent should not be accorded the benefit of the filing date of the earlier application.

The focus on the count, rather than the opponent's claims, is not an accident. The benefit accorded in the specification establishes a constructive reduction to practice for the opponent. In this sense, the notice declaring this interference provisionally rejected Gluckman's corresponding claims under 35 U.S.C. 102(g)(1) in view of the constructive reductions to practice in the Lewis 139 and 913 applications. A constructive reduction to practice exists if there is a single

embodiment within the scope of the count.[24] Cooper v. Goldfarb, 154 F.3d 1321, 1327, 47 USPQ2d 1896, 1901 (Fed. Cir. 1998). This question is significantly different than the question posed under 35 U.S.C. 112[1] (and 35 U.S.C. 119, 120, 121, and 365, which invoke that statute). Under those statutes, compliance requires written description and enabling support for the full scope of the invention, as well as disclosure of any best mode. Cf. Cromlish v. D.Y., 57 USPQ2d 1318, 1319 (BPAI 2000) (questioning whether best mode is even relevant in a constructive reduction to practice determination under Rule 633(g)).

In its motion, Gluckman argues that the Lewis 139 and 913 applications do not support the full scope of the Lewis involved claims. Hence, the motion fails to comply with 37 C.F.R. §1.637(g) and fails to address the right problem. Failure to comply with the rule is a sufficient basis for dismissing or denying the motion.

For completeness, we address the merits of the motion to the extent possible.[25] Gluckman attacks both the written description and the enabling support for the Lewis involved claims. Gluckman alleges that Lewis failed to describe treatment of non-cholinergic neurons. The count, however, includes within its scope the treatment of cholinergic neurons. Hence, even assuming Gluckman's allegation is correct, it falls short of proving that Lewis lacks an embodiment within the scope of the count. Gluckman also alleges that Lewis does not provide

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[24] In this regard, priority is comparable to other anticipation rejections in that a single enabled embodiment suffices to anticipate claims of a broader scope. E.g., Vascath Inc. v. Mahurkar, 935 F.2d 1555, 1562, 19 USPQ2d 1111, 1115 (Fed. Cir. 1991).

[25] The count is the subject matter defined by the disjoint set of two Gluckman claims and two Lewis claims so it is possible to extend Gluckman's arguments to the count somewhat. The Lewis claims constituting the count encompass most of the subject matter of the Gluckman claims constituting the count, so we need not address whether Lewis provided a separate constructive reduction to practice within the scope of the Gluckman claims.

an enabling description for essentially the same reasons provided in Gluckman's preliminary motion 4. Those arguments fail in this context as well for essentially the same reasons.

Upon consideration of Gluckman preliminary motion 7, the Lewis opposition, the Gluckman reply, and the portions of the exhibits the parties cited and discussed, we cannot hold the Lewis 139 and 913 applications insufficient as constructive reductions to practice of an embodiment within the scope of the count. Consequently, Gluckman preliminary motion 7 is DENIED.

VI. Additional benefit for Lewis

Lewis preliminary motion 1 (Paper 37) seeks to have Lewis be accorded the benefit of its 595 application. As explained above, the relevant question is whether the 595 application (or 317 patent) discloses an embodiment within the scope of the count. The 317 patent discloses the use of IGF-1 to treat disease and injury to cholinergic neurons, which is within the scope of the count. Gluckman opposes on the basis that the 317 disclosure (1) is limited to cholinergic neurons rather than the full scope of the Lewis claims, (2) lacks sufficient *in vivo* or relevant *in vitro* examples to enable use of the invention, and (3) does not support post-injury treatment. As previously explained, a cholinergic embodiment is sufficient to establish a constructive reduction to practice of the count. The specification also expressly discloses post-injury treatment of cholinergic neurons. The remaining question is one of enablement.[26]

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[26] For what it is worth, 317 claim 1 appears to be within the scope of the count. In another context, the Federal Circuit has accorded a strong presumption of enablement to patented subject matter. Amgen, Inc. v. Hoechst Marion Roussel, Inc., 314 F.3d 1313, 1354, 65 USPQ2d 1385, 1416 (Fed. Cir. 2003). Whether that holding has any relevance in this context, we do not need to decide because we find sufficient enablement on another basis.

As movant, Lewis bears the ultimate burden to justify the relief it seeks, but Gluckman has the burden of going forward on its contention that the 317 patent does not enable an embodiment within the scope of the count. Nau v. Ohuchida, 1999 Pat. App. LEXIS 15 (BPAI). Gluckman relies on the same enablement arguments that have previously proven unsuccessful. Since the 317 patent has a very similar disclosure, particularly with regard to the induction of ODC, Gluckman's arguments are once again unsuccessful.

Upon consideration of Lewis preliminary motion 1, the Gluckman opposition, and the portions of the exhibits the parties cited and discussed, we hold that the Lewis 595 application provides a constructive reduction to practice of the count.. Consequently, Lewis preliminary motion 1 is GRANTED.

Gluckman has moved (Paper 82) in part to strike Lewis reply 1. Since we have not relied on the Lewis reply, that portion of Gluckman's motion is DISMISSED as moot.

VII. Patentability of Lewis involved claims in view of prior art

Gluckman preliminary motion 2 (Paper 30) seeks judgment that the Lewis involved claims are barred under 35 U.S.C. 102(b) over the Lewis PCT publication.

The Lewis PCT application would, if prior art, anticipate the Lewis claims constituting the count. The disclosure of the Lewis PCT application is substantially the same as the disclosure of the Lewis 595 application. Lewis requested, and in the previous section we granted Lewis, the benefit of the 595 application as a constructive reduction to practice of the count because it contains an embodiment that anticipates the Lewis claims constituting the count. We assume, arguendo, that the other Lewis claims would also be anticipated by the Lewis PCT

application.[27] Hence, the critical question is whether the Lewis PCT is prior art to the involved Lewis claims under § 102(b).

The involved Lewis applications claim the benefit, under 35 U.S.C. 120, of earlier applications including the Lewis 595, 139, and 913 applications. To claim benefit under § 120, the earlier applications must provide support for each claim within the meaning of 35 U.S.C. 112[1]. Tronzo v. Biomet, Inc., 156 F.3d 1154, 1158, 47 USPQ2d 1829, 1832 (Fed. Cir. 1998). To be enabling within the meaning of § 112[1], an application must provide a disclosure commensurate with the scope of the claims. Genentech Inc. v. Novo Nordisk A/S, 108 F.3d 1361, 1365, 42 USPQ2d 1001, 1004 (Fed. Cir. 1997). Gluckman contends that the Lewis PCT is prior art to Lewis because Lewis is not entitled to benefit under § 120 to the intermediate Lewis 139 and 913 applications for the full scope of the Lewis involved claims. Gluckman bases its contention on a lack of enabling support for the involved Lewis claims in the 139 and 913 applications. The fact that we have found support in the Lewis 595 application for a single embodiment within the scope of the Lewis claims constituting the count does not mean that we have found support for the full scope of those claims. As movant, however, Gluckman has the burden of showing that Lewis does not have benefit back through at least its 139 application.

Gluckman's argument for lack of enablement is substantially the same as its argument in its preliminary motion 4, including the failure to address support for each claim separately. This latter flaw is important since each dependent claim should be narrower in scope than its parent

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[27] Appendices E and F of Gluckman's motion 2 "apply" the PCT disclosure to the Lewis involved claims. The motion simply refers the reader to the appendices without further elaboration. The motion is the proper place for the movant's argument. LeVeen v. Edwards, 57 USPQ2d 1406, 1412 (BPAI 2000). By referring to the appendices without further elaboration, Gluckman inappropriately seeks to enlist the Board into making out its argument for it. Rexnord Corp. v. Laitram Corp., 274 F.3d 1336, 1343, 60 USPQ2d 1851, 1855 (Fed. Cir. 2001).

claim and is thus less likely to lack support for its full scope. Gluckman argues that Lewis does not describe the treatment of neurons other than cholinergic neurons and does not provide enabling *in vivo* examples even for cholinergic neurons. The Lewis 139 and 913 applications provide the same relevant disclosure as the involved Lewis applications for the treatment of neurons generally. They also provide the same *in vivo* examples regarding induction of ODC. Consequently, Gluckman's arguments for lack of support sufficient for benefit under § 120 are as unpersuasive as they were in the context of Gluckman's preliminary motion 4.

Upon consideration of Gluckman preliminary motion 2, the Lewis opposition, the Gluckman reply, and the portions of the exhibits the parties cited and discussed, we conclude that Gluckman has not established that Lewis is not entitled to claim benefit under § 120 back through at least the Lewis 139 application and so has failed to establish that the Lewis involved claims are barred under § 102(b) in view of the Lewis PCT application. Consequently, Gluckman preliminary motion 2 is DENIED.

Since Gluckman has not received relief as a result of its preliminary motion 2, the Lewis miscellaneous motion to suppress Gluckman reply 2 and related evidence is DISMISSED as moot.

VIII. Additional benefit for Gluckman

Gluckman preliminary motion 3 (Paper 31) seeks to have Gluckman be accorded the benefit of its New Zealand provisional application. As found above, Gluckman appears to have adequate written description for an embodiment within the Lewis 159 claim 129 alternative of the count. This embodiment is sufficient to accord benefit for the count.

Lewis has two principal points of opposition. Lewis half-heartedly suggests that Gluckman's reported data does not support the embodiment, but notes that the data appears to have been reversed. Given the highly favorable reported outcome, one skilled in the art would have understood that the data was most likely reversed. The second point is that various elements of various alternatives of the count are missing. Assuming this were true, Gluckman need only prove one embodiment within the scope of one alternative of the count, which it has done.

Upon consideration of Gluckman preliminary motion 3, the Lewis opposition, and the portions of the exhibits the parties cited and discussed in the motion and opposition, we conclude that Gluckman has proven that its New Zealand provisional application provides a constructive reduction to practice of an embodiment within the scope of the count. Consequently, Gluckman preliminary motion 3 is GRANTED.

In granting Gluckman's preliminary motion 3, no consideration was given to Gluckman's reply, so the Lewis miscellaneous motion to suppress Gluckman reply 2 and related evidence is DISMISSED as moot.

IX. Patentability of Gluckman involved claims over prior art

Lewis preliminary motion 2 (Paper 38) seeks judgment that Gluckman's involved claims are unpatentable over prior art. Specifically, Lewis contends that Gluckman 460 claims 1-7, 10-13, and 15 and Gluckman 373 claim 1 were barred by the Lewis PCT application. Lewis further contends that Gluckman 460 claims 8 and 9 would have been obvious in view of the combined teachings of the Lewis PCT application and the McMorris or Mozell references. Lewis further notes that if § 102(b) is not available as a bar, the claims would be unpatentable under § 102(a).

Whether the references are § 102(b) references or not depends on whether there is support for Gluckman's claim under 35 U.S.C. 119 in its New Zealand provisional application. Lewis contends that Gluckman did not fully disclose the present invention in its New Zealand provisional application. In particular, Lewis argues that the New Zealand provisional application does not disclose (1) treating neural damage suffered after a CNS insult affecting glia or other non-cholinergic cells (all Gluckman claims), (2) treatment of neural damage where the injury is from Parkinson's disease, multiple sclerosis, or a demyelinating disorder (460 claims 7-9), (3) treatment within 100 hours of injury (460 claim 10), or (4) treatment within 8 hours of injury (460 claim 11).

We must give Gluckman's claims their broadest reasonable construction. In re Zletz, 893 F.2d 319, 321, 13 USPQ2d 1320, 1322 (Fed. Cir. 1989). The broadest reasonable construction of Gluckman's 460 claim 1 and 373 claim 1 is that the treatment must be for a CNS insult "affecting" glia or other non-cholinergic cells, but may also affect cholinergic neurons. We have found support for the treatment of neural damage from a CNS insult affecting tissue comprising non-cholinergic cells. Specifically, the tissues the New Zealand provisional application discloses having primarily treated comprise non-cholinergic neurons (and glia and cholinergic neurons). Hence, the injury to those tissues necessarily "affected glia or other non-cholinergic cells" (as well as cholinergic neurons).

The dependent 460 claims 7-11, however, present a greater problem.[28] The New Zealand provisional application does not teach the specific injuries of claims 7-9. Gluckman does not

[28] Note that a lack of entitlement to benefit under 35 U.S.C. 119 does not, by that fact, render the claims unpatentable under § 112[1]. See Reiffin, 214 F.3d at 1346, 54 USPQ2d at 1918 (an analogous situation under § 120).

offer any direct response other than to note that the New Zealand disclosure shows the efficacy of IGF-1 treatment generally. In the absence of an *in hæc verba* disclosure, Gluckman must provide some explanation of why one skilled in the art would believe Gluckman possessed a treatment for those specific injuries. The best we can infer from Gluckman's opposition is that one skilled in the art might have found such applications obvious. Obviousness, however, is not a basis for written description. Lockwood v. American Airlines, 107 F.3d 1565, 1572, 41 USPQ2d 1961, 1966 (Fed. Cir. 1997).

A similar problem arises for claims 10 and 11, the time-of-administration claims. The New Zealand disclosure teaches two hours. The claims recite up to 8 hours and up to 100 hours. While the claimed ranges certainly encompass the disclosed two hours, their selection appears to be arbitrary in view of the New Zealand disclosure, which provides no basis for suspecting any particular end point beyond two hours. There may be some plausible basis for one skilled in the art to think Gluckman possessed such end points, but Gluckman has not shared that basis with us. Again, absent *in hæc verba* support in the specification, Gluckman must provide at least some explanation.

Gluckman is entitled to the benefit of its New Zealand provisional application for 373 claim 1 and 460 claims 1-6 and 12-15, but not for 460 claims 7-11. The consequence of this holding is that the combination of references that Lewis has proffered are prior art under § 102(b) only with respect to 460 claims 7-11. It is § 102(a) prior art with respect to the other Gluckman involved claims.

A claim is anticipated only when a single prior art reference discloses each and every limitation of the claim. The disclosure need not be express, but may anticipate by inherency

where it would be appreciated by one of ordinary skill in the art. Glaxo, Inc. v. Novopharm, Ltd., 52 F.3d 1043, 1047, 34 USPQ2d 1565, 1567 (Fed. Cir. 1995). The Lewis PCT published application, which is prior art under § 102(a) to Gluckman 460 claim 1 and 373 claim 1, teaches every element of those claims. Specifically, it teaches the treatment of a CNS injury or disease that inherently affects glia or other non-cholinergic cells (as well as treating cholinergic neurons) in a mammal by administering IGF-1 such that it reaches the affected CNS tissue.

Gluckman opposes on the basis that (1) the claims had been allowed over the Lewis PCT published application, (3) the Lewis PCT does not teach *in vivo* protection after the injury has occurred, and (3) the Lewis PCT is limited to treatment of cholinergic neurons. Lewis cannot be estopped from proving anticipation by the fact that an examiner reached a different conclusion (on a different record) in which Lewis did not participate.[29] Moreover, the enabling value of the Lewis disclosure for *in vivo* treatment of existing injury has already been decided above. Gluckman has not urged that its other claims are separately patentable. Hence, they stand, or in this case fall, together. Van Geuns, 988 F.2d at 1186, 26 USPQ2d at 1060.

Upon consideration of Lewis preliminary motion 2, the Gluckman opposition, and the portions of the exhibits the parties cited and discussed, we hold that the involved claims of the Gluckman 460 and 373 patents are anticipated. Consequently, Lewis preliminary motion 1 is GRANTED.

Gluckman has moved (Paper 82) in part to strike Lewis reply 2. Since we have not relied on the Lewis reply, that portion of Gluckman motion is DISMISSED as moot.

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[29] Any more than Gluckman was estopped from challenging a different examiner's ex parte determination (in which Gluckman did not participate) that the claims of Gluckman and Lewis interfere or that examiner's determination that the Lewis claims are fully supported by their specification.

Since Gluckman no longer has any patentable claims corresponding to the count, judgment against Gluckman is appropriate.

Gluckman suggests that because Lewis is not entitled to its § 120 benefit for the full scope of its claims, those claims may be anticipated by Lewis PCT. While such a result is theoretically possible, we have not accepted Gluckman's premise about the lack of enabling support for the full scope of the Lewis claims. A more interesting question is what effect the Mozell article has on the Lewis claims. Note that CNS neuronal survival would be enhanced by preventing demyelination. This question has not been fully developed in this proceeding. We will not reach it now.

X. Lewis inventorship

Lewis has moved under 37 C.F.R. § 1.634 to remove two of its named inventors as inventors in its 159 application. Gluckman opposes. There is no Gluckman motion suggesting that the Lewis claims are invalid for misstating the inventorship. Consequently, decision on this motion is deferred.

ORDER

Upon consideration of the motions of the parties, it is:

ORDERED that Gluckman preliminary motions 3 and 5 be GRANTED;

FURTHER ORDERED that Lewis preliminary motions 1 and 2 be GRANTED;

FURTHER ORDERED that Gluckman preliminary motions 1, 2, 4, 6, 7, and 8 be DENIED;

FURTHER ORDERED that Lewis preliminary motion 3 be DISMISSED as moot;

FURTHER ORDERED that the Gluckman miscellaneous motion to strike Lewis replies 1-3 be DISMISSED as moot;

FURTHER ORDERED that the Lewis miscellaneous motions to suppress Gluckman replies 1-4, 6, and 8, and related evidence, be DISMISSED as moot;

FURTHER ORDERED that the Lewis motion to change inventorship be DEFERRED;

FURTHER ORDERED that the interference be remanded to the administrative patent judge assigned to it for further action not inconsistent with this decision; and

FURTHER ORDERED that a copy of this decision be given a paper number and be entered in the administrative records of the Gluckman 5,714,460 and 5,861,373 patents and of the Lewis 09/064,159 and 09/318,001 applications.

RICHARD TORCZON
Administrative Patent Judge

CAROL A. SPIEGEL
Administrative Patent Judge

SALLY GARDNER LANE
Administrative Patent Judge

BOARD OF PATENT APPEALS AND INTERFERENCES

INTERFERENCE TRIAL SECTION

Notice: Any agreement or understanding between parties to this interference, including any collateral agreements referred to therein, made in connection with or in contemplation of the termination of the interference, shall be in writing and a true copy thereof filed in the United States Patent and Trademark Office before termination of the interference as between said parties to the agreement or understanding. 35 U.S.C. 135(c); 37 C.F.R. § 1.661.

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Paper 116

UNITED STATES PATENT AND TRADEMARK OFFICE
BEFORE THE BOARD OF PATENT APPEALS AND INTERFERENCES

PETER **GLUCKMAN**
and KAROLY NIKOLICS
(5,714,460 and 5,861,373),
Junior Party,

v.

MICHAEL E. **LEWIS**,
JAMES C. KAUER, KEVIN R. SMITH,
KATHLEEN V. CALLISON, and FRANK BALDINO
(09/064,159 and 09/318,001),
Senior Party.

Interference No. 104,553

Before LEE, TORCZON, and NAGUMO, Administrative Patent Judges.

TORCZON, Administrative Patent Judge.

JUDGMENT
(PURSUANT TO 37 CFR §§ 1.640(e) and 1.662(a))

INTRODUCTION

Gluckman was placed under an order to show cause why judgment should not be entered against it (Papers 112 and 114). Gluckman has responded that it does not intend to take further action necessary to have the motions decision reconsidered at the Board or to have a priority period (Paper 115). Consequently, judgment shall be entered against Gluckman.

ORDER

Upon consideration of Gluckman's RESPONSE TO ORDER TO SHOW CAUSE, it is:

ORDERED that judgment on priority as to Count 1 is awarded against junior party Gluckman;

FURTHER ORDERED that Gluckman is not entitled to a patent containing claims 1-15 of the Gluckman 5,714,460 patent, which correspond to Count 1;

FURTHER ORDERED that Gluckman is not entitled to a patent containing claim 1 of the Gluckman 5,861,373 patent, which corresponds to Count 1;

FURTHER ORDERED that the preliminary statements be returned unopened; and

FURTHER ORDERED that a copy of this decision be given a paper number and be entered in the administrative record of the Gluckman 5,714,460 patent; the Gluckman 5,861,373 patent; the Lewis 09/064,159 application; and the Lewis 09/318,001 application.

JAMESON LEE
Administrative Patent Judge

RICHARD TORCZON
Administrative Patent Judge

MARK NAGUMO
Administrative Patent Judge

BOARD OF PATENT APPEALS AND INTERFERENCES

INTERFERENCE TRIAL SECTION

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